GENERAL LIBRARY,
UNIV. OF MICH.
NOV 5 1908

BULLETIN

OF

THE JOHNS HOPKINS HOSPITAL

Entered as Second-Class Matter at the Baltimore, Maryland, Postoffice.

Vol. XIX.—No. 212.] BALTIMORE, NOVEMBER, 1908. [Price, 25 Cents

CONTENTS.

	PAGE
The Volume Curve of the Ventricles in Experimental Mitral Stenosis, and its Relation to Physical Signs. (Illustrated.) By Arthur D. Hirschfelder, M. D.	319
Contributions to the Study of Auricular Fibrillation, Paroxysmal Tachycardia, and the so-called Auriculo-(Atrio-)Ventricular Extrasystoles. (Illustrated.) By Arthur D. Hirschfelder, M. D.	322
Placental Syphilis; A Study of Syphilitic Placentæ with Regard to the Presence of the Spirochæta Pallida. (Illustrated.) By W. O. Pauli, M. D.	326
A Method for Complete Enucleation of the Tonsil. (Illustrated.) By John A. West, M. D.	328
A Simplification of De Grandmont's Operation for Ptosis. By Samuel Theobald, M. D.	331
Three Cases Illustrating the Relationship Between Certain Diseases of the Blood and Sarcoma. By F. G. Bushnell, M. D., D. P. H.	332
The Teaching of Physiological Pathology at the University of Michigan. By F. Peyton Rous, M. D.	336
The Tonsillar Ring and the Paranasal Sinuses as Portals of Entry of Infectious Diseases. By Sylvan Rosenheim, M. D.	338
Notes on New Books	344

THE VOLUME CURVE OF THE VENTRICLES IN EXPERIMENTAL MITRAL STENOSIS, AND ITS RELATION TO PHYSICAL SIGNS.

By Arthur D. Hirschfelder, M. D.,

Associate in Medicine, Johns Hopkins University.

(*From the Medical Clinic of the Johns Hopkins Hospital and University.*)

Method of Experimentation.—The volume curve of the ventricles was obtained by means of Tigerstedt's cardiometers as modified by Yandell Henderson (1), varied only by inverting the recording tambour, as shown in Fig. 1. The carotid pulse curve was recorded simultaneously by means of a Huerthle membrane manometer.

The mitral stenosis was produced by introducing a large curved needle bearing a stout silk ligature backwards through the anterior wall of the left ventricle at a point near the septum and just below the atrio-ventricular groove. This was brought out at a corresponding point on the posterior wall of the ventricle, and then the two ends of the ligature were carried around the atrio-ventricular groove to the outside of the ventricle, so that when they were pulled taut the anterior flap of the mitral valve was pulled laterally, and the orifice thus narrowed. The degree of narrowing could thus be varied at will (Fig. 2).

No curves showing the changes in the volume of the ventricles have been published previously.

In the volume curve of the normal dog's heart, at its usual rate, the upstroke corresponds to the forcing out of the blood into the aorta during systole. This is followed by the beginning of diastole, with the immediate filling of the ventricles (downstroke on curve). The blood enters from the auricles under the force of the venous pressure. The auricles remain passive. In this way about four-fifths of the ventricular filling takes place, the rapidity of filling being represented by the steepness in the descent of the curve. When the rate is as rapid as is usual for the dog's heart, this period of passive filling lasts until the next contraction of the auricle, which drives a small amount of blood (about one-fifth of the total amount) into the ventricle at a rate a little more rapid (curve a little steeper) than the passive filling. The amount which the auricles contribute to the filling of the ventricles, or the systolic output of the auricles never exceeds about one-fourth the systolic output of the ventricles in the normal heart, and frequently falls to one-tenth or less. If the heart rate is slower, that is if diastole is longer, a point is reached at which

the filling of the ventricles comes to a standstill before the auricular contraction sets in. This cessation is due to the fact that an equilibrium is reached between the pressure in veins and auricles tending to drive the blood into the ventricles and the tonus of the ventricular muscle tending to resist further distension. From this point to the time of auricular contraction, the amount of blood entering the ventricles is extremely small, and the volume curve becomes nearly horizontal. Henderson terms this the period of *diastasis*. The transition from diastole proper to diastasis is marked by a more or less definite shoulder upon the volume curve. Henderson states that at this moment the auriculo-ventricular valves close rather suddenly, a phenomenon which can be demonstrated upon a properly constructed model. It can be still more readily demonstrated upon an excised heart from which the upper wall of the auricles is cut away. If water is now poured through the auriculo-ventricular valves from a beaker, it will be noted that the moment the inflow ceases the cusps of the valves flap together, and that the auricular floor formed by them is almost level. The cusps are approximated along the line of closure; and if the height from which the water has been poured exceeds a couple of inches, *the valve is seen to be tightly closed*. The inertia of this closure is quite considerable, requiring a pressure of several centimeters of water to overcome it. The closure is evidently due to the fact that in order that the ventricles may fill to their greatest capacity their cavities must tend to approach the hemispheric form, and the spaces between valve and ventricular wall must be filled as much as possible. This creates a force which tends to float the valves outward, and which, at the moment when inflow ceases, is supplemented by the elasticity of the ventricular walls. It has been suggested that, with the valves even in a normal condition, this closure is sudden enough to produce the third sound of protodiastolic gallop rhythm or the so-called third heart sound (Hirschfelder (2), A. G. Gibson (3), Thayer (4)).

The reason for the change from rapid inflow to stasis is apparently the fact that during the former period the inflow is accelerated by the elastic dilatation of the ventricle and the negative pressure within it, as well as by the large difference between this pressure and the pressure then present in the auricles (from stasis during ventricular systole); whereas after cessation of the rapid inflow and closure of the valves the pressure in the auricles and ventricles is almost exactly equal, and the only force tending to drive blood into the ventricles is that of the blood which accumulates gradually before auricular systole. Since it requires a certain pressure to overcome the inertia of the valves, a period of absolute diastasis without further filling may intervene. If the pressure in the auricles rises more rapidly, or the tonus of the ventricular muscle is low, the filling may proceed further but at a slow rate during the period of diastasis. The further filling of the ventricle from the auricle is the same as when the heart rate is rapid.

In the first stage the volume and systolic output of the ventricles is diminished (volume curve transposed to a higher level), due largely to the fact that the filling of the ventricles is slowed (downstroke upon the curve less steep than normal). The amount of ventricular filling due to the auricles is increased, owing to the accumulation of blood in these chambers at the time they contract, as well as to the fact that the left ventricle is not yet fully distended.

In the second stage the filling of the ventricle proceeds for only a small fraction of the usual period. This is followed by a period during which no further filling occurs until the contraction of the left auricle, which now forces an unusually large amount of blood into the ventricle. This stage corresponds to the condition in which the presystolic murmur is loudest, a fact which is readily demonstrable experimentally.

In the third stage the cardiac volume is much diminished, due mainly to the further diminution in filling, and to the stagnation within the auricle which has led to auricular paralysis. The volume curve is now represented by a short period of systolic output and a short period of ventricular filling, followed by a period during which no further change of volume occurs and no evidence of auricular systole can be seen. The auricle is now paralyzed. This is noted both in animals and in man by the disappearance of the presystolic murmur, and by the appearance in many cases of an "absolute irregularity" in force and rhythm which probably has its origin in the left auricle. This is the so-called "mitralized pulse" (Fig. 5).

The question naturally arises, what relation do these changes in volume bear to the abnormal sounds heard in mitral stenosis, and what light do they throw upon the disturbance of function? The sounds are due to two elements, the muscular contraction and the vibration of the valves. They may also be said to be brought about *actively* during a period of cardiac contraction, or *passively*—during a period of relaxation, the presystolic murmur being quite evidently due to the contraction of the auricle and disappearing when auricular paralysis sets in. Since no other contractions appear between the end of ventricular and the beginning of auricular systole, it is evident that the events occurring within this period are probably of valvular origin.

Study of the volume curve during this period, especially in the second or moderate stage of stenosis, reveals the well-marked elbow which occurs at the end of the period of rapid ventricular filling. This elbow appears also in the normal volume curve of slow hearts, and probably corresponds to occurrence of the so-called third heart sound (protodiastolic gallop rhythm, "bruit de rappel," tá tă tă). As has been stated, there is now good evidence that the mitral valves close passively about this time, and that this diastolic closure of the valves may cause the third sound. It will be noted that according to this explanation of the third sound it is to be considered not an *opening* snap of the auriculo-ventricular valves (as thought by Potain and Sansom (5)) but a *closing* snap due to the diastolic closure. The latter explanation is further borne out by the electrophonographic curves of Einthoven (6) which show that the sound occurs about eighteen hundredths of a second after the second sound at a point at

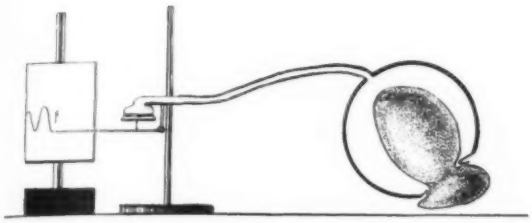

FIG. 1.—Cardiometer and its connection with recording tambour.

FIG. 2.—Cardiometer in place around the auriculo-ventricular groove, showing the position of the ligature when tightened to produce the stenosis.

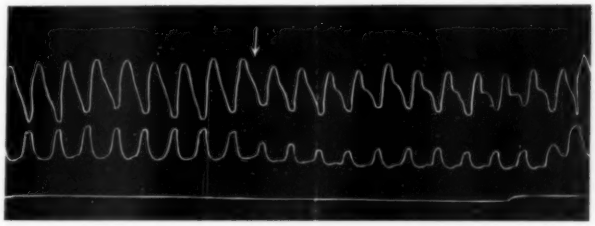

FIG. 3.—Mildest grade of mitral stenosis (produced after the arrow).
Upper curve = volume of the ventricles (upstroke representing systole, downstroke diastole).
Lower curve = carotid pressure tracing with Huerthle membrane manometer.
Lowest line = base line.

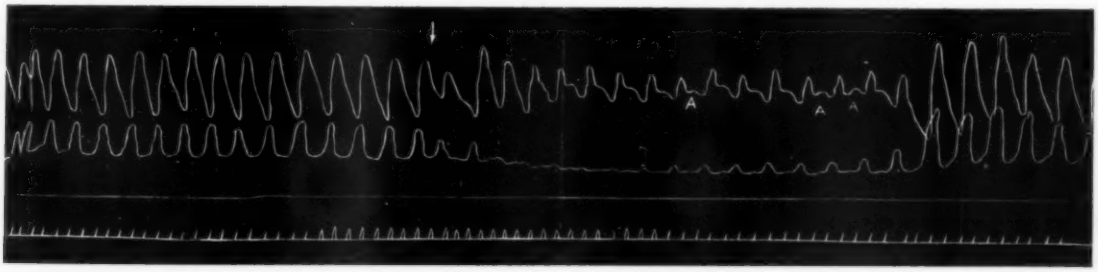

FIG. 4.—More severe grade of stenosis after arrow. Curves as before, showing almost complete disappearance of the auricular wave at *A*, *A*, *A*. Lower line shows time marking in 1/5 seconds.

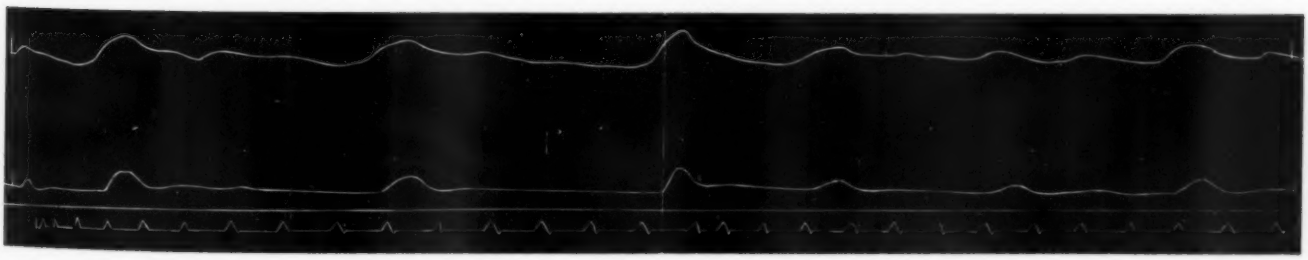

FIG. 5.—Curves as before taken at high speed with very marked degree of mitral stenosis (third stage), showing absence of auricular wave.

which filling has been occurring for a considerable time. Thayer has also shown it to correspond to an interesting wave upon the volume curve which he thinks marks the end of ventricular filling. In mitral stenosis, as has been stated by Thayer, this sound occurs sooner after the second sound than in normal hearts, and in the volume curve in our experiments (compare Fig. 6, C^3 and E^3) it can be seen that the elbow also occurs much earlier in diastole.

Galliverdin (7) has suggested that this sound is due to the shaking of the whole thickened anterior cusp under the influence of the dicrotic recoil above and the inflow from the auricle below. This explanation is possible but might be more readily applied to the early diastolic rumble than to the clear cut "bruit de rappel."

It is quite possible that owing to the variations in the duration of diastole, sounds which appear to be mid-diastolic are really the same as those which with longer diastoles would fall definitely into either the protodiastolic or the presystolic category. At all events the volume curve shows nothing to explain the occurrence of a sound during the period of diastasis.

The volume curve does, however, explain why, as Mackenzie (8) has stated, the sounds produced passively in early diastole are decrescendo in character, while the presystolic sounds are crescendo. The former are produced by the inflow of blood into the ventricle under the influence of the venous pressure, which decreases progressively as the overloaded auricle empties itself. Hence the sounds which it produces decrease. The latter are due to systole of the hypertrophied auricle whose force only gradually reaches its maximum, and hence finds expression in crescendo.

The rare blowing murmur of early diastole, which, as Mackenzie states, is also decrescendo in character, is probably produced by the first passive inflow into the ventricle in the manner usual for blowing murmurs, namely, when the orifice is reduced to a mere slit. In this connection it must be added that it can be readily demonstrated upon the excised dead heart that whenever there is a slight obstruction to inflow from the auricle and the inflow takes place at low pressure, the cusps of the mitral valve remain approximated along the greater part of the line of closure, and are separated only along a small portion of their extent. In this way the functional orifice is much smaller than the actual orifice, and the *relative* stenosis adds itself to the organic stenosis. A narrowing of the channel sufficient to give rise to a blowing murmur might thus readily occur and be transitory in character, a fact which would correspond to the transitory character of such blowing murmurs observed clinically (Bard). Certain it is that the blowing diastolic murmur heard at the apex, in the left axilla and back, but not over the aortic or pulmonic area, deserves more attention as an occasional physical sign in mitral stenosis than is usually accorded to it.

In passing, it must be added that it can be readily demonstrated upon the excised heart that when a certain pressure is maintained in the ventricle, and when this pressure approxi-

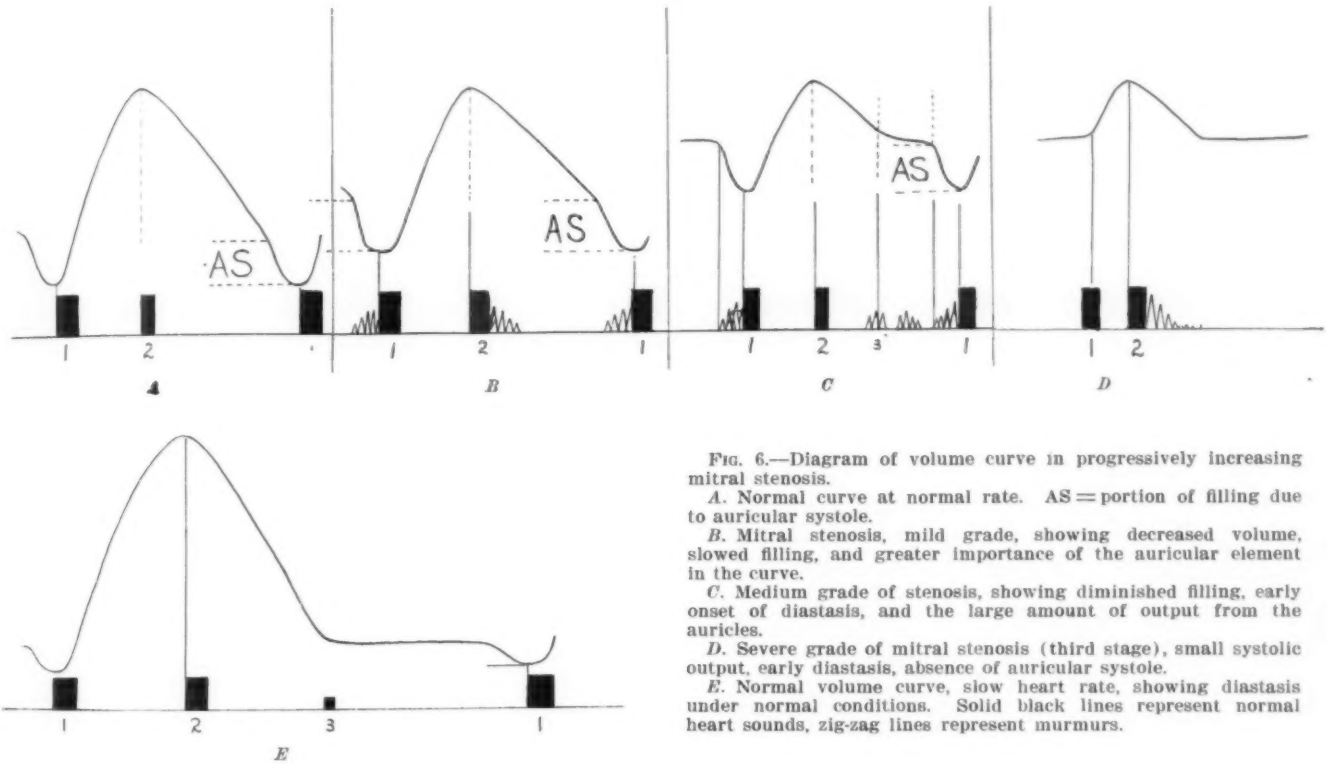

FIG. 6.—Diagram of volume curve in progressively increasing mitral stenosis.

A. Normal curve at normal rate. AS = portion of filling due to auricular systole.

B. Mitral stenosis, mild grade, showing decreased volume, slowed filling, and greater importance of the auricular element in the curve.

C. Medium grade of stenosis, showing diminished filling, early onset of diastasis, and the large amount of output from the auricles.

D. Severe grade of mitral stenosis (third stage), small systolic output, early diastasis, absence of auricular systole.

E. Normal volume curve, slow heart rate, showing diastasis under normal conditions. Solid black lines represent normal heart sounds, zig-zag lines represent murmurs.

mates the pressure in the auricle, the mitral cusps no longer open to their full extent, but the opening is reduced to a small space near the middle of the cusps. A functional mitral stenosis is produced. When the auricular pressure is further increased or the pressure within the ventricle diminished, the cusps again open along their whole line of closure.

The existence of such a functional mitral stenosis in aortic insufficiency is assumed by Bard (l. c.), though he gives no actual demonstration of its production. He believes that this functional mitral stenosis is responsible for the presystolic rumble (Flint murmur) of aortic insufficiency, a theory which would be in perfect accord with the above-mentioned experiment.

Upon the limitation of the thrill and murmur of mitral stenosis to a small area about the apex, one of my experiments seems to throw some light. In a dog whose tricuspid valve was slightly incompetent, owing to some sclerotic thickening along the greater part of the line of closure, a systolic thrill could be felt along the wall of the right auricle, but only over a line about three millimeters wide exactly opposite the point where the leak was taking place. At no other points could the thrill be felt. This thrill was therefore due to the direct impact of the regurgitant stream against the wall of the auricle, and not to any low pitched vibrations of the wall as a whole. It seems probable that localized thrills and also murmurs may arise thus at the points where the narrow stream strikes against the heart wall. It can easily be demonstrated that the stream passing through the narrowed mitral orifice is just such a narrow stream, whose direction is, as a rule, toward the apex. In aortic insufficiency the impact of the stream would be diminished by the pressure encountered within the ventricle, and the thrill produced would be, as is observed clinically, rarer and less intense than that of mitral stenosis. Moreover, the intensity of the thrill would be reduced proportionately more than the murmur, so that the latter would remain after the former had disappeared.

I desire to express my thanks to Mr. Julian M. Wolfsohn for his valuable assistance in the execution of these studies, and to the authorities in charge of the Hunterian Laboratory of the Johns Hopkins University for the privilege of pursuing them there.

BIBLIOGRAPHY.

1. Henderson, Yandell (with the collaboration of M. McR. Scarborough and F. P. Chillingworth): The volume curve of the ventricles of the mammalian heart, and the significance of this curve in respect to the mechanics of the heart-beat and the filling of the ventricles. Am. J. Physiol., Boston, 1906, XVI, 325.

2. Hirschfelder, A. D.: Some variations in the form of the venous pulse. Johns Hopkins Hosp. Bull., Balt., 1907, XVIII, 265.

3. Gibson, G. A. Upon the significance of a hitherto undescribed wave upon the venous pulse. Lancet, Lond., 1907, II, 1380.

4. Thayer, W. S.: On the early diastolic heart sound (the so-called third heart sound). Bost. M. and S. J., 1908, CLVIII, 713.

5. Potain and Sansom (quoted from L. Bard): Die physikalische Zeichen der Mitralstenose. Samml. klin. Vortr., Leipz., 1907, No. 137.

6. Einthoven, W.: Ein dritter Herzton. Arch. f. d. ges. Physiol., Bonn, 1907, CXX, 31.

7. Galliverdin, L.: Nouvelle explication du bruit de dédoublement du rétrécissement mitral par le claquement diastolique de la mitrale. Lyon médical, 1905, CV, 401.

8. Mackenzie, James: The extrasystole, etc. Quart. J. M., Oxford, 1907, I.

CONTRIBUTIONS TO THE STUDY OF AURICULAR FIBRILLATION, PAROXYSMAL TACHYCARDIA, AND THE SO-CALLED AURICULO-(ATRIO-)VENTRICULAR EXTRASYSTOLES.

By Arthur D. Hirschfelder, M. D.,
Associate in Medicine, Johns Hopkins University.

(From the Medical Clinic of the Johns Hopkins Hospital and University.)

Ludwig and Hoffa (1), Einbrodt (2), Sigmund Mayer (3), Vulpian (4), Neumann (5), Fischel (6), and Trendelenburg (7) have investigated the effects of direct electrical stimulation of the mammalian heart, and have found that weak stimuli, either faradic or galvanic, bring about a sudden increase in rate in the chamber stimulated, while strong stimuli throw it into a state of fibrillation. If the electrodes be applied to the auricle alone, the tachycardia is communicated to the ventricles as well, and the rate of the entire heart is increased. The peculiarity of this increase lies in the fact that it is described by some of these writers as a doubling of the pulse rate, a description which was later applied by August Hofmann (8) to the change of rate ushering in the attacks of paroxysmal tachycardia. Similar sudden changes in rate have also been recorded by v. Ziemmsen (9) during galvanization of a human heart which was separated from the electrodes by only the skin and pericardium. As is readily seen from a study of their curves and from the results obtained by repeating their experiments, this so-called doubling is not an exact one, but ranges from 1.6 to 2.1 : 1 instead of exactly 2 : 1.

If the ordinary electrodes are applied to any part of the auricles, or fish-hook electrodes are inserted into the auricular wall, and a weak faradic stimulus applied, no change of rate is observed. If the stimulus is increased, auricular contractions suddenly increase from 1.6 to 2.1 times the previous rate, each auricular being followed by a ventricular contraction. The conduction time is usually lengthened when the

stimulus is a comparatively weak one (Fig. 1). The rate is usually regular, and both rate and conduction time return to normal at once when the stimulation ceases.

Occasionally the rhythm of both auricles and ventricles is irregular, sometimes interrupted by pauses. The rhythm of the ventricles is more irregular than that of the auricles, owing to the diminution in both conductivity and contractility. Fischel has shown, moreover, that when the right ventricle is dilated after an intravenous salt infusion, its contractions manifest an irregularity in force, while the left ventricle, which is not subjected to the strain, remains regular. When the intensity of the stimulus to the auricles is augmented, the irritability of the heart seems to be greatly increased. The tachycardia begins as before, with a rate about the same as when weak stimuli are employed, but its duration outlasts the period of stimulation (Fig. 2). The return to the normal rate occurs spontaneously, suddenly, and without warning. The conduction time during the tachycardia is diminished, but returns to normal at the normal rate. Between attacks the heart rate may be quite irregular, being interrupted by typical auricular extrasystoles, with normal or lengthened conductivity, shortened bigemini (shortened compensatory pause), and also by other extrasystoles of a peculiar type, which will be discussed in a subsequent paragraph.

When the intensity of the faradic stimulation is still further increased (the coil being pushed up as far as possible, and two dry cells used in the primary circuit), the contractions of the auricles assume a less co-ordinated character. The rate increases very greatly and contractions become shallow, irregular and difficult to record. At a certain stage they pass into a state of typical fibrillation (Fig. 3). However, the rapidity is so great and so irregular and the contractions so shallow that it is impossible to determine exactly the point at which co-ordinated contraction of the entire chamber disappears. The rate of the ventricle as a rule is not affected by the transition to auricular fibrillation, but continues, as before, at approximately twice the normal. Occasionally, and especially when the heart is very irritable, the onset of auricular fibrillation brings about fibrillation of the ventricles as well, after which the dog's heart does not usually recover.[1]

The intensity and duration of these changes seem to bear a definite relation to the irritability of the heart as well as to the strength of stimulus. In some hearts, especially in those of strong dogs which had not been subjected to prolonged operations, prolonged fibrillation set in after faradization of one- or two-fifths of a second. In others it required much longer and more intense stimulation to produce the same effect. However, it was observed usually that if several short stimulations succeeded one another at short intervals they exerted a cumulative effect, that of the last being by far the greatest and most prolonged. In other words, the irritability, rhythmicity and conductivity seemed to be increased by the faradization, but the effect upon irritability seemed to outlast that upon either rhythmicity or conductivity, since both of these properties return to normal during the intervals between tachycardias, while the response to a given stimulus is increased. Conductivity is not affected during these intervals (since the conduction time is then normal), but it varies with the changes in rhythmicity, as stated above.

The fibrillation of the auricles sometimes ceases after vagus stimulation, and the rate of the whole heart returns to normal (Fig. 4); sometimes its effect is only momentary and the fibrillation and tachycardia are resumed (Fig. 5).

The changes which accompany faradization of the ventricles resemble those of faradization of the auricles in many ways. When the stimulus is a weak one there is no response; when it is moderately intense the ventricular rate suddenly increases, exactly as is the case with the auricle when that chamber is stimulated. The rate is nearly but not exactly double the normal, and commences and subsides within one or two cardiac cycles. The auricle does not respond to the ventricular rhythm, but continues at its own original rate. When the stimulus is further increased, fibrillation of the ventricles occurs, and the cardiac action becomes paralyzed unless it be revived by stimulation of the vagus (Garrey).

Considerable interest attaches itself to the peculiar extrasystoles by which the interval between auricular and ventricular contraction is shortened, in some cases even to the point of almost absolute synchronism between the two chambers. This condition was first studied by James Mackenzie (11), who described it as "Inception of the Rhythm of the Heart by the Ventricles" and thought that it was due to overexcitation of the fibers joining the auricles and ventricles (fibers of the His bundle). As he thought that they arose in the nodal portion of this bundle, he termed them "nodal extrasystoles." Hering and Rihl (12) reached the same conception a year later, designating them as "atrio-ventricular extrasystoles," and state that this diagnosis can be made whenever the a - c interval accompanying the extrasystole is shorter than that with the preceding regular beat. These opinions, however, are based entirely upon *a priori* reasoning and not upon laboratory experiment. Lehmann, the only observer who has published experiments upon stimulating the bundle directly, did so by stimulating all the tissue within the heart-block clamp, and thus stimulated both auricle and ventricle directly even more than the His bundle. Needless to say such evidence is of no value.

On the other hand, in our experiments there was no stimulus applied to the His bundle or to any portion of the cardiac musculature in the vicinity of the sinus or conducting system. The stimulation affected the auricular appendix directly and it alone (Fig. 6). We are, therefore, confronted with two possible explanations of the occurrence of these extrasystoles: Either they are to be regarded as representing merely a special type of auricular extrasystole in which increase in conductivity is associated with increase in irritability; or it is possible that when an abnormal stimulus is applied to any part of an auricle whose irritability is markedly increased, the impulse may spread at once to the cells of the conduction system

[1] Garrey (10) has shown that peculiar sudden increases of rate follow chemical stimulation of the cardiac nerves in dogs.

and stimulate them to autonomic contraction and to a series of abnormal contractions such as were observed by Gaskell (13) to follow stimulation of the corresponding fibers in the heart of cold-blooded animals. The effect upon these cells, though a secondary phenomenon, might overshadow the effect upon the auricles.[2]

The conditions in our experiments were, therefore, not quite analogous to those in Mackenzie's cases, in which such extrasystoles occurred. In these cases degeneration, arteriosclerosis and fibrous myocarditis within the bundle of His were revealed at autopsy, and Mackenzie believes that these changes acted as irritating lesions upon that structure. It seems highly probable that this explanation applies to such cases.

In our experiments, however, no changes in the His bundle were present. On the other hand, in a very large number of experiments in which hæmorrhages into the His bundle and its vicinity were produced by the introduction of the Erlanger clamp, I have never seen any such extrasystoles occur singly or in groups, nor do they ever occur during the tightening of the clamp.

Whichever explanation may be correct, it is certain from the above experiments that extrasystoles with shortened conduction time may arise as a result of stimuli applied directly to the auricle, and need not necessarily indicate the presence of an irritative lesion within the bundle of His or even within the system of Purkinje fibers, *i. e.*, in the muscular path between the coronary sinus, the septum membranaceum and its intramuscular and subendocardial ramifications in the ventricles, as claimed by Mackenzie.

The clinical significance of the above-mentioned experiments lies in a certain parallelism between the phenomena observed and attacks of paroxysmal tachycardia in man. The conceptions of the latter condition are not very clearly defined. It seems certain at present that we must differentiate sharply between those cases in which the rate gradually increases and does not reach its maximum for a minute or two, and those in which the rate becomes approximately doubled, trebled or quadrupled within one or two cardiac cycles.

The former group is common in neurotic individuals, convalescents from typhoid fever and other infectious diseases, and hyperthyroidism, and represents simply an exaggeration of the normal changes of rate under the influence of emotion, excitement, etc. That is, the tonic activity of the vagus diminishes, and the influence of the accelerators predominates during the periods of tachycardia, while the balance is restored when the rate returns to normal.

The cases in which the onset and cessation of tachycardia are so sudden that they occur within one or two cardiac cycles represent the group which may properly be designated as idiopathic paroxysmal tachycardia. As stated above, August Hofmann (l. c.) called attention to the fact that the change in rate represented an almost exact sudden doubling or quadrupling, with subsequent sudden return to the preceding rate. Trebling of the rate has also been described, but doubling is most common. However, in most of the cases reported the ratios have not been absolutely mathematical, and such changes as from 80 to 140 (1 : 1.7) are seemingly more common than the exact doubling.

Hofmann ascribed the change in rate to the existence of a sino-auricular heart block (2 : 1 rhythm) which passed off during the paroxysm, so that the auricles and ventricles then beat at the same rate as the sinus, while in the interim they were responding to only alternate impulses from the sinus. Although there would seem to be no *a priori* objection to this explanation, no cases have been reported which demonstrate its clinical existence, the nearest approach being a case published by me (14) but which is susceptible of a different classification.

That the disturbance in these cases with sudden changes of rate is not merely a disturbance in the physiological antagonism of vagi and accelerators was proved by Gerhardt (15), and by myself, since it was possible to paralyze the vagi of our patients with atropin without bringing on an attack. I further showed that the stimulation of the unrestrained accelerators due to mild exercise undertaken while the effect of atropin was at its height, was also insufficient to bring on an attack.

The reported cases, accompanied by venous tracings, which alone are worthy of record, seem to fall into two distinct groups. First, those characterized by the presence, during the attack, of a wave due to auricular contraction before each ventricular contraction. The attack subsides by exact halving of the rate, though the rate of the auricle persists. There is evidently a partial heart-block (2 : 1 rhythm) between the auricles and the ventricles, associated with an abnormally high auricular rate. This latter factor may in itself bring about fatigue of the His bundle, whose occasional recovery causes the return to the full rate, the onset of the paroxysmal tachycardia. A considerable number of cases belonging to this group have been reported especially by Hofmann (l. c.) Gerhardt (l. c.), Rihl (16), and Schmoll (17).

Second, those cases in which the venous pulse is of the ventricular type during the attack, of the normal auricular type between attacks, and where no signs of atrio-ventricular block (2 : 1 rhythm) are ever present. On the other hand, after the attacks there may occur an irregularity due to the presence of extrasystoles with shortened conduction time. Cases of this group have been described by Mackenzie, myself, and others.

It should be noted that these attacks of paroxysmal tachycardia very closely resemble the experimental tachycardias of auricular origin which were described in the first part of this paper. The wave due to auricular contraction disappears, as might be expected, if the auricles are in a state of fibrillation or their contractions become too weak to give a wave upon the venous tracing. The pulse rate rises suddenly to approxi-

[2] In favor of this view must be mentioned the fact that in certain extrasystoles as in No. 1, Fig. 6, the effect upon the ventricle is much more evident than upon the auricle. Either the auricular extrasystole is here absent or the extrasystole is too feeble to be recorded. Nevertheless, the *original* stimulus for these extrasystoles affected the auricle and not the His bundle or the sinus, to which it might have been communicated.

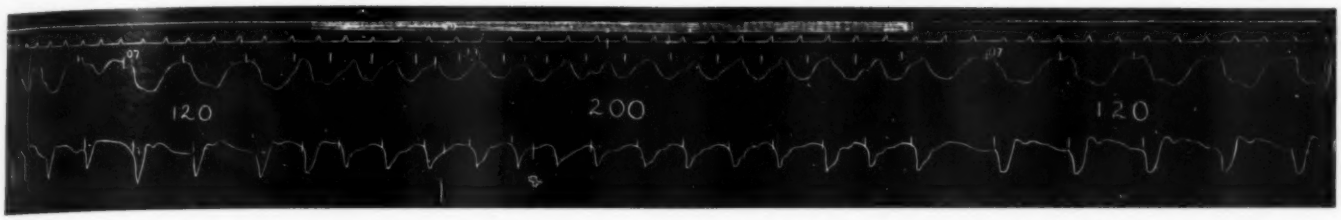

FIG. 1.—Weak faradization of right auricle. The figure shows a heart with rate of 120 per minute, and conduction time .07 second. During stimulation the rate rises suddenly to 200, the conduction time to .13 second. On cessation of stimulation both fall suddenly to normal.

Upper line = stimulation.
Second line = time in 1/5 second.
Third line = contractions of right ventricle (down-strokes).
Fourth line = contractions of right auricles.

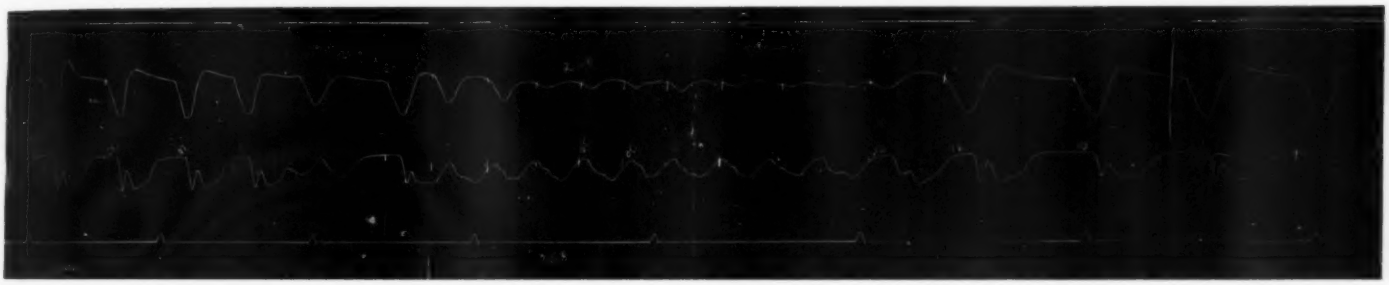

FIG. 2.—Faradization of right auricle with strong stimulus repeated at short intervals. Heart rate rises from 126 to 258 per minute during a period which outlasts the stimulus; and falls spontaneously to 120 after about 2½ seconds. Conduction time falls from .09 to .06 during the tachycardia; rises to .10 after rate becomes slow.

Upper line = stimulation of auricle.
Second line = auricular contractions.
Third line = auricular contractions.
Lowest line = time in seconds.

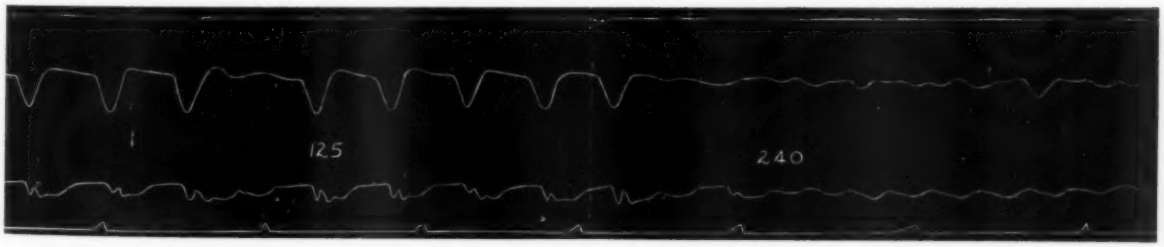

FIG. 3.—Onset of auricular fibrillation after short faradic stimulation of an irritable auricle. Order of curves as in Fig. 2. Fibrillation of auricles and regular tachycardia persisted for several minutes.

FIG. 4.—Cessation of auricular fibrillation under the influence of maximal stimulation of the vagus. Fall of rate from 240 to 140. Upper line represents stimulation of the vagus: curves otherwise as in Figs. 2 and 3.

FIG. 5.—Same as Fig. 4, except that the heart was more irritable and resumed the rapid rate as soon as the vagus stimulation passed off.

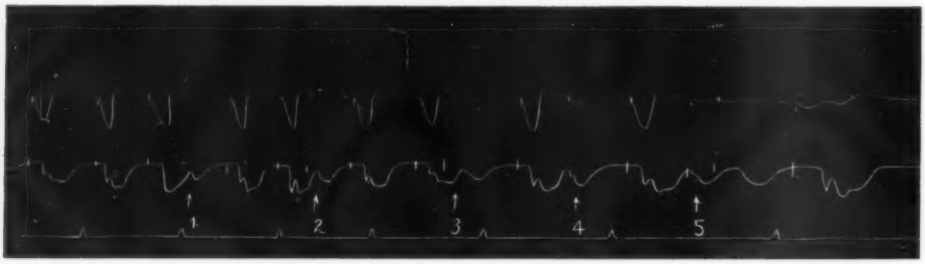

FIG. 6.—Direct faradization of right auricular appendix. Extrasystoles with normal conduction time (1, 2, and 3) and also with shortened conduction time (4 and 5); almost synchronous in auricles and ventricles

mately double the normal. The mild attacks cease upon vagus stimulation, the severe ones continue in spite of it. After an attack has subsided, extrasystoles with shortened conductivity may be present. This condition has further many points in common with the paroxysmal irregularity which Cushny and Edmunds (18) described in some clinical cases, and also in dogs whose auricles manifested "spontaneous" fibrillation, and perhaps also with the sudden changes in rate which Garrey has observed to follow the chemical stimulation of cardiac nerves.

Moreover, Reid Hunt (19), and Cushny and Edmunds have seen the auricles of the dog pass into a state of fibrillation after cessation of vagus stimulation, although Garrey states that neither he nor Hewlett, who was working in his laboratory, had ever seen this occur unless the heart was already in a state of greatly increased irritability from the concomitant action of some other factor. My own experience tends to confirm this opinion.

A large number of cases have, moreover, been reported in which lesions of the central nervous system were found at autopsy, and not infrequently lesions involving the nucleus or fibers of the vagus (Reinhold, Hofmann, Pitres, Oppenheim (20), Schlesinger (21), or adhesions along the course of the vago-sympathetic (Pal). Occasionally the condition has been seen in association with epilepsy (Nothnagel, Schlesinger), in early tabes (Hirschberg), in multiple sclerosis (Müller), and in exophthalmic goiter (Hoesslin and Brown).

The question arises as to how these cases shall be classified. None of the paroxysms reported in these cases has been studied from the physiological standpoint, and their mode of onset and subsidence has not been considered. In the study of cases of exophthalmic goiter and hyperthyroidism under my observation, and of the charts of such cases from American and German clinics, I have seen none in which the pulse rate did not rise and fall gradually. The condition here is, therefore, merely an instability in the equilibrium between vagi and accelerators, and must be differentiated from the attacks typical of paroxysmal tachycardia, at least until all doubts as to the pathological physiology of the latter have been dispelled. For the present all classifications should be based upon only those cases which have been studied by means of venous tracings, if possible accompanied by autopsy findings, as has been done by Mackenzie and Keith. Investigations with the electrocardiogram will unquestionably throw much light upon the condition. Classifications based upon mere clinical associations without reference to the mode of onset or nature of the paroxysm, as has been attempted by Schmoll, can lead only to confusion in the present state of our knowledge.

These observations, taken in connection with the experiments mentioned above, throw doubt upon the theory first advanced by Mackenzie and subsequently advocated by Schmoll, that the lesion or disturbance which brings on the true paroxysmal tachycardia lies in the bundle of His or in the Purkinje cells of the conduction system, though this may apply to occasional cases. It seems more likely that *true paroxysmal tachycardia is usually caused by some one of a number of conditions which bring about a condition of increased irritability of the heart muscle*, especially of the auricles, which probably pass into a state of fibrillation. Just as in the animal a slight stimulus suffices to throw the heart into paroxysms of tachycardia under these conditions, so in man the act of defecation, slight emotional excitement, or sudden awakening from sleep represent stimuli which have been known to precipitate attacks.

The underlying condition of increased irritability of the heart muscle may be brought about either by some direct irritant lesion in the muscle itself, and especially in the auricular muscle or the Purkinje fiber system, or through the medium of the cardiac nerves. From the experiments it would appear that the frequent repetition or duration of action of the stimulus are fully as important as its intensity, and hence it need not be surprising that in man as in animal little evidence of the increased cardiac irritability or of the stimuli producing it can be seen between the attacks.

In conclusion I desire to express my thanks to the authorities in charge of the Hunterian Laboratory of the Johns Hopkins University for the privilege of pursuing these studies there.

BIBLIOGRAPHY.

1. Ludwig and Hoffa: Einige neue Versuche ueber Herzbewegung. Ztschr. f. rationelle Med., 1849, IX, 128.

2. Einbrodt: Ueber Herzreizung und ihr Verhältniss zum Blutdruck. Sitzber. d. k. Akad. d. Wissensch., Wien, 1859, XXXVIII, 345.

3. Mayer, Sigmund: Ueber die direkte elektrische Reizung des Säugethierherzens. Ibid, 1873, LXVIII, 3te Abtheil., 1.

4. Vulpian, A.: Note sur les effets de la faradisation directe des ventricules du cœur chez le chien. Arch. de physiol., Paris, 1874, I, 975.

5. Neumann, R.: Untersuchungen ueber die Wirkung galvanischer Ströme auf das Frosch- und Säugethierherz. Arch. f. d. ges. Physiol., Bonn, 1886, XXXIX, 403.

6. Fischel, R.: Ueber Tonusänderungen und die anderen graphisch an den vier Abtheilungen des Säugethierherzens bei elektrischer Reizung desselben zu ermittelnden Erscheinungen. Arch. f. exper. Path. u. Pharmakol., Leipz., 1897, XXXVIII, 228.

7. Trendelenburg, W.: Untersuchungen ueber das Verhalten des Herzmuskels bei rhythmischer elektrischer Reizung. Arch. f. Physiol., Leipz., 1903, 271.

8. Hofmann, Aug.: Neue Beobachtungen ueber Herzjagen. Deutsches Arch. f. klin. Med., Leipz., 1903, LXXVIII, 39.

9. v. Ziemmsen: Studien ueber die Bewegungsvorgänge am menschlichen Herzen, sowie ueber mechanische und elektrische Erregbarkeit des Herzens, u. s. w. Ibid, 1882, XXX, 270.

10. Garrey, W. E.: Effect of chemicals on the heart nerves. Calif. State M. J., San Francisco, 1907.

———. Some effects of cardiac nerves upon ventricular fibrillation. Am. J. Physiol., Bost., 1908, XXI, 283.

11. Mackenzie, James: On the inception of the rhythm of the heart by the ventricles. Brit. M. J., 1904, I, 529.

———. New methods in the study of affections of the heart. Ibid, 1905, I, 813.

———. Abnormal inception of the cardiac rhythm. Quart. J. M., Oxford, 1907, I, 39.

———. The extrasystole: A contribution to the functional pathology of the primitive cardiac tissue. Ibid, 1908, I, 182 and 481.

12. Hering, H. E. and Rihl, J.: Ueber atrioventriculäre extrasystolen. Ztschr. f. exper. Pathol. u. Therap., Berl., 1906, II, 510.

———. Experimentelle Untersuchungen ueber Herzunregelmässigkeiten an Affen. Ibid., 1906, II, 525.

13. Gaskell, W. H.: The properties of cardiac muscle. Schaefer's Text-Book of Physiology, Lond., 1900, Vol. II.

14. Hirschfelder, A. D.: Observations upon paroxysmal tachycardia. Johns Hopkins Hosp. Bull., Balt., 1906, XVII, 337.

15. Gerhardt, D.: Beiträge zur Lehre von den Extrasystolen. Deutsches Arch. f. klin. Med., Leipz., 1905, LXXXII, 509.

16. Rihl, J.: Analyse von fünf Fällen von Ueberleitungstörungen. Ztschr. f. exper. Pathol. u. Therap., Berl., 1905, II, 83.

17. Schmoll, E.: Paroxysmal tachycardia. Am. J. M. Sci., Phila., 1907, CXXXIV, 662.

18. Cushny, A. R. and Edmunds, C. W.: Paroxysmal irregularity of the heart and auricular fibrillation. Ibid, 1907, CXXXIII, 66.

19. Hunt, Reid: Direct and reflex acceleration of the mammalian heart. Am. J. Physiol., Boston, 1899, II, 395.

20. Reinhold, H.: Beiträge zur Pathogenese der paroxysmalen Tachykardie. Ztschr. f. klin. Med., Berl., 1908, LIX, 168.

21. Schlesinger, H.: Ueber paroxysmale Tachykardie, u. s. w. Samml. klin. Vortr. innerer Med., No. 131, Leipz., 1906.

The complete bibliography of cases will be found in the articles of Reinhold, Schlesinger, and Schmoll.

PLACENTAL SYPHILIS; A STUDY OF SYPHILITIC PLACENTÆ WITH REGARD TO THE PRESENCE OF THE SPIROCHÆTA PALLIDA.

By W. O. Pauli, M. D.,

Medical House Officer, Johns Hopkins Hospital.

Since the discovery of the characteristic micro-organism of syphilis by Schaudinn and Hoffmann in 1905, various authors have reported positive findings of *Spirochæta pallida* in the placentæ of syphilitic infants; while, on the other hand, other investigators have searched for them with repeated negative results, but at the same time were able in the majority of cases to demonstrate the organisms in the fœtal organs. In view of these contradictory reports, the following work was undertaken at the suggestion of Prof. J. W. Williams.

A study was made of the syphilitic cases which presented themselves for treatment in the Obstetrical Department of the Johns Hopkins Hospital, in order to determine if possible, in what percentage of cases the *Spirochæta pallida* is demonstrable in placentæ presenting histological evidence of syphilis. This would afford a ready means of making a positive diagnosis of syphilis and thus enable the physician to institute proper prophylactic treatment against repeated miscarriages in subsequent pregnancies, and to place the child upon immediate specific treatment. Then, too, it is frequently stated in the literature, that it is doubtful whether the changes found on microscopical examination of the placenta are characteristic manifestations of syphilis or not, as the changes which are ordinarily so considered, may also be found in conditions not syphilitic. Thus, Mohn and others state that placental syphilis cannot be diagnosed without demonstrating the *Spirochæta pallida*. This, however, seems to be too drastic a postulate, for in a great majority of the cases, in which the gross and microscopical examination of the placenta reveals the characteristic features of syphilis, the *Spirochæta pallida* can be demonstrated in the fœtal organs, although none can be found in the placenta. As will be shown later, this affords sufficient evidence to make a positive diagnosis of syphilis of the placenta.

On gross examination, the characteristic signs of syphilis of the placenta are: (1) A relative increase in the size and weight of the placenta as compared to that of the child; the normal ratio being 1 to 6, while in syphilis it often increases and becomes 1 to 4 or even more. (2) The maternal surface instead of being dark reddish in color becomes pale pink, and has a greasy feel. (3) The fissures which mark off the cotyledons, are deep and wide in syphilis, as compared with the normal.

Examination of a teased specimen of the fresh placenta will often enable one to differentiate suspicious cases by careful examination of the villi. The procedure adopted in the routine examination of all placentæ in the Obstetrical Laboratory is to take a minute portion from the material surface of the placenta by means of a pair of forceps and then hold it

Micro-photograph of *Spirochæta pallida* in Fœtal Liver Tissue.

on a slide under the tap, so as to spread apart the branches of the villi and to wash away any blood clot; after which it is examined under the microscope. In syphilis, the branching is less delicate than normal, the ends of the finer branches are thickened and club-shaped; the blood vessels are usually absent in the finer branches and only poorly seen in the main villous stems, while the stroma is more opaque and presents a granular appearance.

On examining hardened sections of the placenta which have been stained with hæmatoxylin and eosin, the picture is almost typical. The short thickened branches of the villi occupy the entire field, obliterating the interspaces, the stroma is dense and the nuclei of the connective tissue cells appear

in great numbers, while the blood vessels are obliterated, due to an endarteritis and only few red blood cells are seen in the vessels of the larger villi.

During the 12 months extending from July, 1907, to July, 1908, 825 placentæ were examined, and among these there were 25 diagnosed as syphilitic after microscopic examination of sections by Prof. J. W. Williams. Autopsies were obtained on 16 out of 25 infants, in 14 of which an anatomical diagnosis of congenital syphilis was made. Such a diagnosis was based upon the increase in size and weight of the liver and spleen, the presence of pneumonia alba in the lungs, and of definite osteochondritis at the epiphyseal line of the long bones. The anatomical diagnosis was confirmed by the demonstration of the *Spirochæta pallida* in 11 cases, while five others proved negative.

The placenta was examined for the *Spirochæta pallida* in 24 of the 25 cases; in one case the tissue was thrown away by mistake, but positive results were obtained in no case, in spite of careful and repeated search. The Levaditi method of impregnating the tissue with silver nitrate and reducing by pyrogallic acid solution was used in every case. The procedure is as follows: thin blocks of tissue are hardened in 10 per cent formalin for 24 to 48 hours, then transferred directly to 95 per cent alcohol for 24 hours and washed in distilled water. When the tissue sinks in the water the blocks are transferred to brown bottles containing 1.5 per cent solution of silver nitrate and placed in the thermostat at 37° C. for three days. They are then rinsed in distilled water and immersed in 4 per cent solution of pyrogallic acid containing 4 per cent formalin. After remaining in the thermostat for 24 hours longer, until reduction has taken place, it is removed to room temperature and washed, dehydrated, and cleared in aniline oil and xylol, then mounted in paraffin. The sections are cut as thin as possible, usually 3 to 5μ, and mounted without a counter stain and examined under the oil immersion lens of the microscope. Sections were cut from only one block of tissue, excepting in two cases in which blocks were taken from two different portions of the placenta. Although careful search was repeatedly made, in not a single instance was it possible to demonstrate a typical *Spirochæta pallida* in the placenta, while they were found in large numbers in the fœtal organs in 11 cases.

A summary of the 24 cases is given in the accompanying table.

Upon reviewing the literature on the subject it is found, that a few isolated cases have been reported, in which the *Spirochæta pallida* was demonstrated in the placenta, but in these cases the number of organisms were usually very limited, and long and tedious search was necessary in order to

No.	Age.	Color.	Marital.	Parity.	Luetic history.	Period of gestation.	Length of fœtus.	Condition.	Anat. diag.	Weight of fœtus.	Weight of placenta.	Relative prop. wts.	Mic. exam. placenta.	*S. pallida* in placenta.	*S. pallida* in fœtal organs.
										Gms.	Gms.				
5588	17	Wh.	S.	0p.	June, 1907.	9 mos.	45 cm.	Still born.	Cong. lues.	2920	1020	1 : 3	Syphilis.	Negative.	Positive.
5687	19	Bl.	S.	Ip.	Denied.	7 "	37 "	Macerated.	No autopsy.	640	250	1 : 3	"	"	.....
5758	17	Bl.	M.	0p.	Denied.	6 "	...	Macerated.	No autopsy.	920	350	1 : 3	"	"	....
5796	21	Bl.	S.	Ip.	Denied.	9 "	50 "	Macerated.	Negative.	2730	510	1 : 5	"	"	.....
5845	17	Bl.	S.	Ip.	Denied.	8 "	....	Macerated.	No autopsy.	....	370	....	"	"	.....
5868	25	Bl.	M.	IIp.	Denied, 1 miscarriage.	4 "	18 "	Not macerated.	No autopsy.	90	160	2 : 1	"	"	.....
5884	20	Wh.	S.	0p.	Dec., 1907.	7 "	36 "	Macerated.	Cong. lues.	1670	580	1 : 3	"	"	Positive.
5896	18	Bl.	M.	Ip.	Denied.	9 "	48 "	Macerated.	No autopsy.	3150	590	1 : 5.3	"	"	.....
5929	15	Wh.	S.	0p.	Denied.	9 "	46 "	Pemphigus, 4 days.	Cong. lues.	2895	650	1 : 4	"	"	Positive.
5944	19	Wh.	S.	0p.	Denied.	8 "	39 "	Macerated.	Cong. lues.	1780	910	1 : 2	"	"	Positive.
5971	34	Wh.	M.	IIp.	Denied, 5 miscarriages.	9 "	46 "	Macerated.	Cong. lues.	2600	800	1 : 3.2	"	"	Negative.
5975	19	Bl.	M.	IIp.	Denied.	7 "	34 "	Lived 5 days.	No autopsy.	1575	600	1 : 2.6	"	"	.....
6000	18	Bl.	S.	0p.	Denied.	7 "	39 "	Lived 2 days.	No autopsy.	900	260	1 : 3.5	"	"	.....
6026	20	Bl.	M.	VIIp.	Denied, 2 miscarriages.	8 "	40 "	Still born.	No autopsy.	1350	340	1 : 4	"	"	.....
6170	18	Wh.	M.	0p.	Denied, 1 miscarriage.	9 "	45 "	Macerated.	Cong. lues.	2250	560	1 : 4	"	"	Positive.
6174	24	Bl.	M.	Ip.	Feb., 1908.	7 "	34 "	Macerated.	Cong. lues.	1050	280	1 : 3.8	"	"	Positive.
6175	32	Bl.	M.	Ip.	Aug., 1908.	9 "	45 "	Macerated.	Cong. lues.	2150	480	1 : 4.5	"	"	Positive.
6204	19	Bl.	M.	Ip.	Denied.	9 "	46 "	Macerated.	Cong. lues.	2320	580	1 : 4	"	"	Positive.
6210	24	Bl.	S.	Ip.	Denied.	5 "	25 "	Still born.	No autopsy.	440	210	1 : 2	"	"	.....
6224	26	Bl.	M.	IIp.	Jan., 1905.	9 "	47 "	Macerated.	Cong. lues.	1800	400	1 : 4.5	"	"	Positive.
6230	24	Wh.	M.	0p.	Feb., 1908.	7 "	35 "	Macerated.	Cong. lues.	1920	470	1 : 2	"	"	Positive.
6297	16	Bl.	S.	0p.	Denied.	5 "	Twins { 24 cm. No. 1 / 25 " No. 2	Macerated.	1. Cong. lues. 2. Cong. lues.	290 / 300	430	1 : 1.4	"	"	1. Neg. 2. Neg.
6310	20	Bl.	S.	Ip.	Mar., 1908.	8 "	41 cm.	Macerated.	Cong. lues.	1510	420	1 : 3.6	"	"	Positive.
6356	17	Bl.	M.	0p.	June, 1908.	8 "	41 "	Lived 12 hours.	Cong. lues.	1500	500	1 : 3	"	"	Negative.

find them, so that Baum, in his text-book of Obstetrics states that the *Spirochæta pallida* is practically never found in the placenta.

Including the various individual cases reported in the literature, I have been able to collect 120 syphilitic placentæ which were examined for the *Spirochæta pallida* with positive findings in 16 cases.

	No. of placentæ examined.	*S. pallida* found.
Paaschen	5	5 cases.
Versé	2	0 case.
Schultz	2	1 "
Menetrier and Duval	1	1 "
Wallich and Levaditi	13	1 "
Huebschmann	1	1 "
Mohn	16	6 cases.
Bab	39	2 " doubtful.
Tissier and Girauld.	17	1 *S. pallida.*

L. Tissier and Girauld examined 40 cases of suspected syphilis, occurring during the course of three months in 323 admissions at the Maternity Division of the St. Louis Hospital at Paris. In the series there were 65 abortions, 9 of which presented signs of syphilis; 21 deliveries of a premature macerated fœtus; 3 deliveries at term of living infants of a doubtful nature. The placentæ were examined in 17 of the 40 cases and they found a single *Spirochæta pallida* in one instance. They also were able to demonstrate the *Spirochæta pallida* in the fœtal organs of 10 cases which were over six months' gestation, and emphasize the fact that they were unable to find any *Spirochæta pallida* in the organs of infants under six months' gestation.

Hans Bab, working in Professor Baum's clinic in Berlin, examined 39 syphilitic placentæ of which 37 proved negative and in 2 cases the findings were doubtful. He states that in not a single instance did he obtain a perfectly definite *Spirochæta pallida.* Felix Mohn on the other hand was more successful. He was able to demonstrate the *Spirochæta pallida* in 6 out of 16 syphilitic placentæ, but generally they were very few in number and at times only a single spirillum was detected. Moreover, where the *Spirochæta pallida* had been demonstrated in the placenta, as in the cases reported by Wallich and Levaditi, as well as by Menetrier and Rubens Duval, marked manifestations of congenital syphilis, such as pemphigus, were present in the fœtus.

The conclusions to be drawn from this study of the subject are (1), that the *Spirochæta pallida* is rarely found in syphilitic placentæ, and then only after long and tedious search. (2) That the anatomical changes observed in the placenta, are the result of toxins produced by the *Spirochæta pallida* in the fœtal organs and not to the immediate effect of the organisms upon the villi themselves. (3) The placenta is not the nidus of infection. The fact that the *Spirochæta pallida* is never found in the maternal portion of the placenta, but only in the blood vessels and stroma of the fœtal villi, would suggest that the placenta offers greater resistance to the invasion of the organisms, which is probably due to the steady flow of antibodies or immune substances from the mother through the maternal circulation.

In closing, I wish to thank Prof. J. W. Williams for the privilege of using the material in the Obstetrical Department and for the interest he has shown in the progress of the work.

LITERATURE.

1. Bumm: Grundriss zum Studium der Geburtshülfe, 4th ed., 1907, p. 316.

2. Paaschen: Sitzungsbericht d. Aerztlichen Vereins zu Hamburg. Münch. med. Wchnschr., 1905, No. 49.

3. Versé: Die Spirochæte pallida in ihren Beziehungen zu den syphilitischen Gewebsveränderungen. Münch. med. Wchnschr., 1906, No. 29, p. 1440.

4. Schultz: Distribution of Treponema pallidum. J. Med. Research, Vol. X, 1906.

5. Menetrier and Rubens Duval: Sur un cas de septicémie à spirochètes de Schaudinn chez un nouveau-né syphilitique. Bull. etmém. Soc. méd. des hôpitaux, Séance du 29 décembre, 1905, No. 38.

6. Wallich and Levaditi: Recherches sur la syphilis du placenta. Ann. de gynéc. et d'obst., February, 1906.

7. Huebschmann: Spirochæte pallida (Schaudinn) und Organerkrankung bei Syphilis congenita. Berl. klin. Wchnschr., 1906, No. 24.

8. Mohn: Die Veränderungen an Placenta, Nabelschnur und Eihaüten bei Syphilis und ihre Beziehungen zur Spirochæte pallida. Ztschr. für Geburtsh. und Gynäk. 1907, No. 59, p. 263.

9. Bab: Bakteriologie und Biologie der kongenitalen Syphilis. Ztschr. für Geburtsh. und Gynäk., 1907, No. 60, p. 161.

10. Tissier and Girauld: Congenital Syphilis. Bull. etmém. Soc. obst. de Paris, 1908, No. 1.

A METHOD FOR COMPLETE ENUCLEATION OF THE TONSIL.

By John A. West, M. D.,

Assistant Laryngologist, Out-patient Department, Johns Hopkins Hospital.

Owing to observations which have been made by internists and surgeons, as well as by laryngologists, showing the connection between the tonsils, as a portal of infection, and such conditions as rheumatism, endocarditis, enlarged glands of the neck, recurring peritonsillitis, etc., the complete enucleation of the tonsil has become an important operation, and it is for the laryngologist to develop as perfect a technic as possible for this operation.

Aside from minor details of little importance, the various methods so far described may be classed as follows: (1) Galvano-caustic dissection, advocated by Pynchon. Owing to the reaction likely to follow, this method finds few adherents. (2) Enucleation with the snare or ecraseur after having freed the tonsil from the pillars as recommended by Casselberry. It is claimed for this method that it is followed by less hemorrhage. This has been denied, and certain it is

that severe bleeding may follow the use of the snare; moreover, wounds made by snaring through tissues are, as a rule, followed by more reaction and do not heal as kindly as the clean cut wound of the scalpel. (3) Dissection by cutting the tonsil loose from the anterior pillar first, and then proceeding backwards. This is sometimes done with scissors (Robertson), or the operation may be begun with a knife and scissors, and finished with a snare (Goodale), or beginning by freeing the tonsil from the anterior pillar and proceeding backwards, the operation may in many instances be completed with a straight knife alone (Ballenger). This method of Ballenger, as will be seen further on, is open to objection only because at the last stage of the operation the hemorrhage may be so great that it is unsafe to continue with the knife. In view of this danger the operators using the anterior method usually complete the operation with a snare or ecraseur.

During the past year at the Johns Hopkins Hospital, with the encouragement of Dr. Mackenzie, who very kindly placed the material of the clinic at my disposal, I have been able to perfect a method which is different from any so far described, and which I have found to be not only entirely satisfactory, but preferable. Instead of attacking the tonsil from the anterior pillar first, it has been found of distinct advantage to free the posterior pillar from the tonsil as the first step in the dissection. On the surgical principle that clean cut wounds heal best, it is preferable to complete the operation with knives to the exclusion of the snare or ecraseur.

The method of procedure is as follows:

PREPARATION.

It is well to have the patient gargle the throat with some antiseptic solution, e. g., hydrogen peroxide or potassium permanganate, and if the crypts of the tonsil are filled with cheesy material, they should be washed out with a syringe.

Local is much to be preferred to general anæsthesia, as the hemorrhage is controlled more easily with the former, and, moreover, with local anæsthesia the patient himself can hold down the tongue with a depressor, which is often of assistance. Schleich infiltration is found to do excellently, about 10-20 cc. being injected at different points around the tonsil after swabbing the entire area of mucous membrane with a weak cocaine solution. With very nervous patients a hypodermic of morphia will help considerably.

Instruments, sponges, etc., must of course be sterilized.

OPERATION.

The operation may be divided into four steps:

I. The tonsil is grasped and pulled forwards with a pair of toothed forceps, and with an L-shaped knife (Fig. 7*a*), completely separated from the posterior pillar (Fig. 1).

II. Holding the tonsil again with the forceps and exerting traction towards the median line, with a straight knife (Fig. 7*b*), begin below the tonsil posteriorly at the point where the first incision ends, and make a circular cut around the tonsil meeting the first incision again at its upper end (Fig. 2). No attention is paid to the Plica triangularis.

III. Pulling the tonsil with the forceps well out into the mouth, and continuing with the same knife (Fig. 7*b*), begin above and posteriorly to cut through the loose connective tissue between the capsule of the tonsil and the superior constrictor muscle (Fig. 3). When there is little hemorrhage the dissection can readily be completed with this knife alone.

IV. When the hemorrhage is considerable and the field of operation thereby obscured, it is preferable after dissecting the tonsil perhaps three-quarters free, to complete the operation with a blunt-pointed knife (Fig. 7*c*), by entering the blade into the incision made below the tonsil (Step II), and

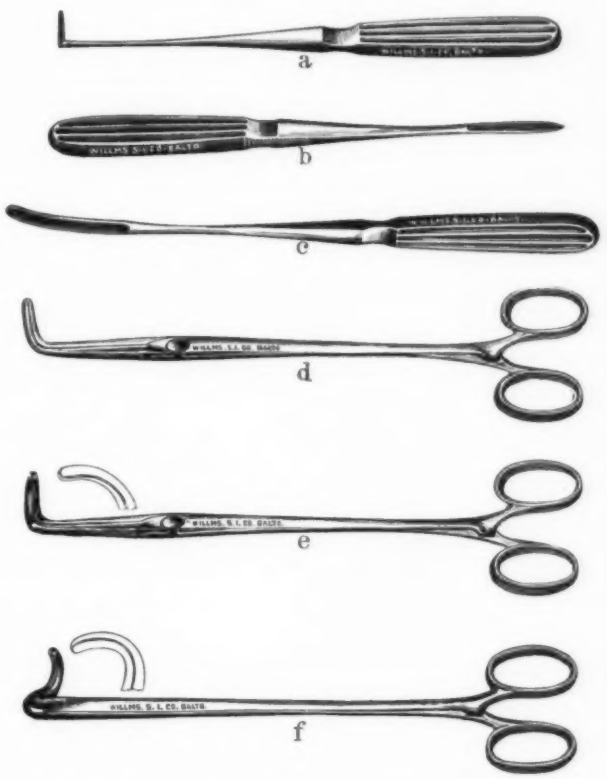

FIG. 7.—*a*. L-shaped knife for dissecting tonsil from posterior pillar in Step I of operation, as shown in Fig. 1.

b. Straight knife used in Steps II and III of operation (Figs. 2 and 3).

c. Blunt-ended knife for completing the enucleation as described in Step IV of operation and shown in Fig. 4.

d and *e*. Hemostatic forceps for clamping bleeding areas on the posterior and lateral part of the Sinus tonsillaris.

f. Hemostatic forceps for clamping bleeding areas on the posterior surface of anterior pillar.

cutting upwards (Fig. 4) to meet the incision (Step III), thus completely enucleating the tonsil with its capsule.

The three knives shown in diagram, having rather stout handles, which prevent turning in the hand, serve admirably for the work.

Time.

While the time actually occupied in enucleating a tonsil by this method is usually less than two minutes, the time from beginning to end of the dissection varies greatly, depending largely upon the patient. Where the throat is very sensitive the operation may be prolonged considerably, owing to the fact that retching may interfere with making the incisions accurately, and hence it is necessary to watch one's chance, so to speak, and to incise between the attacks of retching or gagging.

As the removal of both tonsils at one sitting is frequently quite a shock to the patient, it is often better, particularly in adults, to remove them at an interval of several days, unless the patient remains over night in the hospital, in which case both can be enucleated with safety at one sitting.

Control of Hemorrhage.

There is little danger from bleeding if the operator has had experience in controlling tonsillar hemorrhage, and if he is careful to stop *all* bleeding before leaving the patient. It is wrong to leave the patient when the hemorrhage has been controlled but partially, for oozing may continue for hours and a great deal of blood may be lost. The application of pressure and hemostatic clamps is to be preferred to such hemostatic drugs as the preparations of iron. When the latter are used a large clot of blood usually forms in the Sinus tonsillaris, from behind which a steady stream of blood, at times not at all easily discovered, may be running down the pharynx. Patients have bled to death in a few hours where this method has been used.

The following procedure for controlling the hemorrhage has been found entirely satisfactory and practically eliminates the danger. At the moment of completing the enucleation of the tonsil the bleeding is usually brisk, and a plug of sterile gauze is at once pressed tightly into the cavity between the pillars of the fauces, with an ordinary pair of nasal forceps. This usually stops most of the smaller vessels within a few minutes. Should the hemorrhage continue a long time, the bleeding areas are caught with specially constructed clamps, as the ordinary surgical hemostatic forceps are not well adapted to clamping in the Sinus tonsillaris behind the anterior pillar. The clamps (Figs. 7*d* and *e*) are suited for holding bleeding areas on the posterior and lateral part of the cavity, while the clamp (Fig. 7*f*) is intended for the posterior surface of the anterior pillar. It is more convenient to have two sets of these clamps to fit the right and left sides of the throat. By inverting them, however, one set can be made to fit into the Sinus tonsillaris of either side. The clamps are left *in situ* for about half an hour, and if on removing them the hemorrhage begins anew, they are replaced and allowed to remain for two hours. These clamps are more effective than the Mikulicz model and cause the patient less discomfort. If they are used, it should never be necessary to sew the pillars together.

Comparison of the Anterior and Posterior Methods of Operating.

Were it not for the hemorrhage generally met with, it would be a matter of no consequence whether the dissection were begun at the anterior or the posterior edge of the tonsil. But owing to the bleeding, which is usually quite brisk, it is decidedly advantageous to begin the operation posteriorly for the following reasons:

If one follows the anterior method of Ballenger, towards the end of the operation the tonsil will be found attached to the posterior pillar (Fig. 5), and sometimes also in the region of the tongue. In cases of severe hemorrhage it is very difficult, if not quite impossible, to see the line of junction of the posterior pillar with the tonsil; as the effusion of blood may be so great as to leave the upper surface of the tonsil alone visible. In such cases to proceed further with the knife is dangerous; the operation has to be completed with a snare or ecraseur, and even then there is danger of snaring off part of the posterior pillar.

The posterior method has been found of particular value in the cases of profuse hemorrhage, as by this procedure it is always possible to complete the enucleation with only the knives. When the posterior method is used, towards the end of the operation the tonsil will be found attached only by a certain amount of loose tissue to the fascia covering the superior constrictor muscle of the pharnyx at some point (shown schematically by the shaded area in Fig. 6), near the middle of the Sinus tonsillaris. This remaining pedicle is easily severed with the blunt-ended knife, as described in Step IV of the operation, without any danger whatever of injury to the posterior pillar, no matter how great the hemorrhage may be.

In a word, the last step of the anterior operation consists in cutting the tonsil from the posterior pillar, and the region of the tongue, which owing to the abundant hemorrhage may be difficult to do accurately. Should the posterior pillar by mistake be removed, the resulting cicatricial tissue, by restricting the movement of the velum palatinum may give the patient a nasal voice. On the other hand, the final step in the posterior operation consists in severing the tonsil from its connection at a point somewhere near the middle of the Sinus tonsillaris, which is easily done regardless of the amount of hemorrhage, and with no danger whatever to the posterior pillar, that had been dissected free at the beginning of the operation.

It will thus be seen that the procedure here described possesses two distinctive features: (1) The dissection of the tonsil beginning at the posterior edge and proceeding anteriorly. (2) The control of the hemorrhage by specially constructed clamps.

Although it is certain that skilful operators will have good results by following any of the well-known procedures, we believe the method here described to be the safest, quickest, and least uncomfortable for the patient.

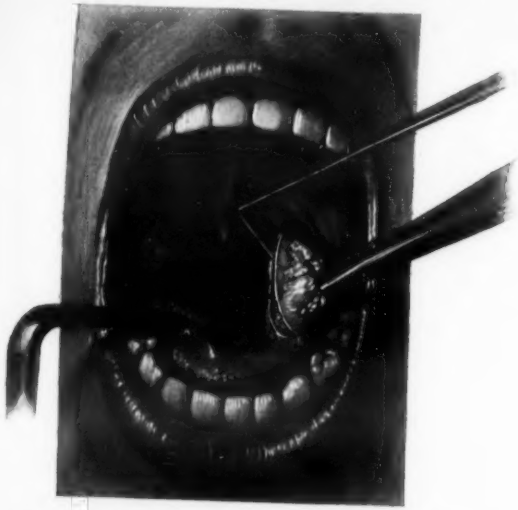

FIG. 1.—First step of operation. The dissection of the tonsil from the posterior pillar.

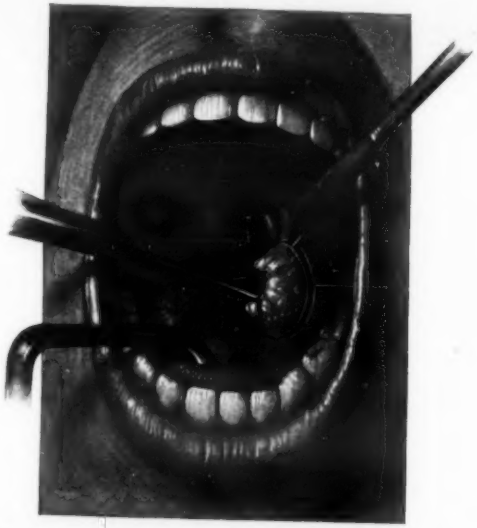

FIG. 2.—Second step of operation. A circular cut is made anteriorly around the tonsil through the mucous membrane thus permitting the tonsil to be drawn well out into the mouth.

FIG. 3.—Third step of operation. The loose tissue connecting the capsule of the tonsils to the fascia of the superior constrictor muscle is cut through the greater part of its extent from behind forwards and from above downwards.

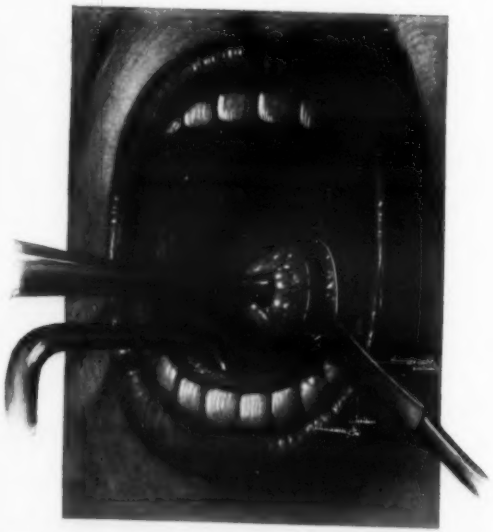

FIG. 4.—Fourth step of operation. The capsule of the tonsil is severed from its remaining attachment to the fascia of the superior constrictor muscle, thus completing the enucleation. The position of the pedicle cut through is shown schematically by the shaded area in Fig. 6.

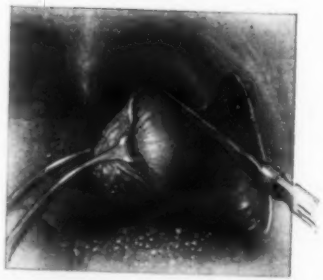

FIG. 5.—Ballenger's figure modified from his recent book showing the final step of his operation.

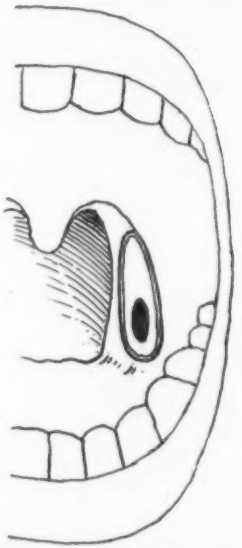

FIG. 6.—Showing schematically the position of the last bit of tissue connecting the capsule of the tonsil with the fascia of the superior constrictor muscle, which is severed in the fourth step of the operation as shown in Fig. 4.

A SIMPLIFICATION OF DE GRANDMONT'S OPERATION FOR PTOSIS.[1]

By SAMUEL THEOBALD, M. D.,

Clinical Professor of Ophthalmology and Otology, Johns Hopkins University; Ophthalmic and Aural Surgeon to the Johns Hopkins Hospital.

Not long since there came under my care at the Johns Hopkins Hospital a case of acquired, bilateral ptosis, in which the lid fault—itself but one of many manifestations of a grave, general myasthenia—was decidedly more marked upon the left than upon the right side.

As there was little ground to hope for improvement from non-surgical measures, I operated upon the more affected, left, eye by the method of de Grandmont as modified by Gruening, and, with a view of obtaining as perfect a cosmetic result as possible, I decided to wait for a few days and see the effect of this first operation before proceeding with the second eye.

Meanwhile, I considered how I might correct most exactly this slighter defect of the right lid; and after some reflection it occurred to me that this could be accomplished best by everting the lid and, without any disturbance of the external integument, removing directly from the tarsal cartilage—the outlines of which would then be easily seen—a strip of such width as might seem to be indicated, the excision to include, of course, the overlying conjunctiva. This procedure, furthermore, commended itself as involving less traumatism, and as being altogether a simpler and more easily performed operation than that of de Grandmont.

After waiting three days, I proceeded to operate upon the right eye in accordance with the plan determined upon. The eye having been first anæsthetized by the instillation of a 10 per cent solution of cocaine, the upper lid was everted and a few drops of a 2 per cent solution of cocaine, to which had been added a small quantity of a 1:1000 suprarenalin solution, was injected into and, perhaps, beneath the cartilage at several points along the line of the proposed incisions. An incision, begun with the knife and completed with scissors, was then made through the conjunctiva and the whole length of the cartilage parallel with, and about 3½ mm. from, the face border of the lid. In similar fashion, a second incision was made *parallel with the first* and about 3 mm. from it, and the included strip of cartilage and conjunctiva was excised. The edges of the divided cartilage were next brought accurately together by means of three fine, black silk sutures, and, in less than half the time required to execute the de Grandmont procedure upon the other eye, the operation was completed. Furthermore, when the lid was turned back into its normal position, one could see at a glance exactly to what extent the ptosis had been corrected, the elevation of the lid-margin being practically the same as was found a few days later when the œdema consequent upon the incision had disappeared. The anæsthesia induced was complete, and there was no complaint, whatever, of pain. Probably a weaker solution of cocaine would have been equally effective, but the house surgeon, who looked after this feature of the operation, did not care to take this chance. Snellen's lid-clamp might possibly have facilitated the making of the incision through the cartilage, but it did not occur to me to use it.

I have emphasized the point that the second incision through the cartilage was made *parallel with the first*. This, again, is a departure from the de Grandmont operation; and it seemed to me to possess an important advantage, in that both edges of the divided cartilage are straight—not one straight and the other concave as in his procedure—and therefore more apt to unite evenly and without distortion. Furthermore, it seemed that the removal of a broader strip from the middle of the cartilage could have only the effect of elevating disproportionately the center of the lid-margin. The result proved the correctness of this reasoning—the lid was raised squarely throughout its length.

It was feared that the stitches upon the conjunctival aspect of the lid—which, as a matter of precaution, were not removed until the fourth day—might cause the patient considerable annoyance, but this did not prove to be the case.

To summarize the advantages which the procedure I have outlined seems to possess, it may be said: That it is a simpler operation, involving considerably less traumatism, than that of de Grandmont, and consequently may be performed in half the time; that it does away with the buried stitches which he employs, or the deep ones, coming out on the lid-margin, suggested by Gruening; that the parallel incisions through the cartilage are preferable to those made by de Grandmont for the reasons just set forth; that one may judge at once exactly the effect produced in elevating the lid, and may with little trouble increase this effect, if it seems desirable to do so; and that the restoration of the parts to their normal condition occurs in much less time than in either the de Grandmont or Gruening operation.

The simplicity and advantages of the plan which had suggested itself of attacking the cartilage from the inner surface of the lid seemed so obvious that I could not but ask myself whether this method had not occurred to others. A perusal of de Grandmont's description of his operation[2] showed, at all events, that it had not been thought of by him. Turning next to the account of Bowman's operation,[3] which differs from de Grandmont's in that the upper lip of the cartilage, with a portion—in the case described "about half an inch"—of the tendon of the levator palpebræ, is removed. I was interested to learn that in his first case (the one just referred to) he

[1] A paper read before the American Ophthalmological Society, July 16, 1908.

[2] "J. de Méd. de Paris," 1891, p. 296.

[3] "Royal London Ophthalmic Hospital Reports," Vol. I, p. 34.

operated from the conjunctival surface of the lid, though in subsequent cases he went through the external integument; which is not surprising, as he doubtless found it easier to resect the levator tendon by this route.

Since this paper was written, I have received from the patient's physician, Dr. G. F. Bush, of Carnesville, Ga., a letter in reply to one which I wrote him asking for information as to the further progress of the case. Writing on July 13, the operation on the left eye having been performed May 22, and that on the right eye May 25, he says: "The operation on the left eye is not a success. The lid remains œdematous and heavy . . . . The weight seems to produce a dropping or ptosis. The right eye is all O. K. The result is excellent. You can not detect that it has been operated upon."

THREE CASES ILLUSTRATING THE RELATIONSHIP BETWEEN CERTAIN DISEASES OF THE BLOOD AND SARCOMA.

By F. G. Bushnell, M. D., D. P. H.,

Pathologist Stephen Ralli Memorial Pathological Department, Sussex County Hospital, Brighton, England.

Case I.—Leukanæmia (or atypical lymphocytic leukæmia with a leukæmic or pseudoleukæmic blood condition).

A. W., 20 years, male. Admitted to the Stephen Ralli Memorial Hospital, July 5, '07, under the care of Dr. E. Hobhouse, for "anæmia."

Previous Health.—Has never been laid up before; being always healthy.

Personal History.—Unmarried; is a laborer by occupation. For many years has taken two or three pints of beer daily, but never spirits.

Present Illness.—Commenced gradually about 10 weeks ago, with a feeling of sleepiness and weariness, making him unfit for work. These feelings have gradually increased and he took to bed five weeks ago. He has had no other complaint. He attributes the illness to extra work, three weeks previously he having worked eleven in place of eight hours a day.

On Admission.—Temperature 98.4°; pulse 112, dicrotic; respiration 28. He is very white and looks ill. Conjunctivæ and gums pale. Pupils widely dilated, equal and active. Breath short, and even speaking causes dyspnœa. No œdema; no clubbing of fingers; no enlarged glands.

Circulatory System.—Pulse 112, large, soft and dicrotic; regular. Heart apex-beat forcible in fourth space, just inside nipple (3½ inches from mid-line). Left border in left nipple-line. Right border one inch to right of mid-sternum. Upper border, third rib.

Sounds: "Bruit de diable" present. First sound followed by soft systolic murmur all over precordia with loud pulmonary second sound. Capillary pulsation present. The carotids and other vessels pulsate forcibly.

Respiratory System.—Healthy.

Abdomen.—Distended by enormous enlargement of liver.

Liver.—Upper border, fourth rib; lower border, anterior superior iliac spine. Surface smooth, firm and slightly tender; margin quite sharp.

Spleen.—Very large, filling up Traube's space; anterior border, sixth rib, upper margin; upper border, level of nipple; left border, two fingers' breadth below costal margin. No moveable dullness in abdomen. No other mass felt.

Urine.—Acid, 1017. No albumen, no sugar, no blood; heavy deposit of urates.

August 10, '07. After the removal of blood from the ear for examination there has been continuous oozing; slight oozing from gums; has had general aching all over for a few days.

August 13, '07. Epistaxis occurred.

August 17, '07. Legs and feet, especially left, slightly œdematous. Much pain in knees—sleeping badly.

August 28, '07. General condition better. Liver edge two inches below costal margin. Spleen not felt—œdema of feet almost gone.

September 8, '07. Slight epistaxis and bleeding from gums four days ago.

September 10, '07. Bleeding from ear after removal of blood. Slight oozing from gums. Not so well. P. 126; R. 28; no pain.

Post-Mortem Examination.

The autopsy was made October 11, '07, by Mr. A. W. Anderson, M. B. Body very pale, well nourished, soft. Rigor mortis barely present; post-mortem lividity absent.

Lungs.—Pleuræ normal; no fluid present.

Right Lung.—Two patches of consolidation the size of a large marble in upper lobe, œdematous.

Left Lung.—Very œdematous all over.

Heart.—Pericardium contained about six or eight ounces of clear serous fluid; no thickening of pericardium or inflammation. Heart very large, right ventricle dilated, much ante-mortem clot in both auricles. Numerous minute hæmorrhagic spots underneath pericardial and endocardial surfaces, well marked about auriculo-ventricular valves. Heart muscle flabby and shows "tabby cat" striations of fatty degeneration on endocardial surface most marked on papillary muscles. Valves showed no abnormality. Aorta healthy.

Liver.—Generally and enormously enlarged, weight 4750 grm. The cut surface showed some fatty and fibrous changes. A faint Prussian blue reaction was obtained irregularly, apparently about interstitial tissue.

Spleen weighed 1775 grm., surface dark red, substance firm. A spleniculus was attached to upper border of tail of pancreas.

Kidneys.—Left, 475 grm. weight; right, 250 grm. Both showed extensive fatty changes, cortex and pyramids being indistinguishable; surface pale without striation; capsules stripped easily.

Pancreas.—No abnormality.

Mesenteric Glands.—All somewhat enlarged, red and soft.

Bone Marrow.—Right femur very dark-red and firm.

Stomach.—No naked eye change.

Other organs not examined.

Bacteriological Examination.

Cultures made from marrow and spleen were sterile.

Morbid Histology.

Liver shows no iron reaction in sections. The capillaries are stuffed with large mononuclear cells. There are nucleated red cells (megaloblasts).

Marrow shows large mononuclear cells in great numbers, with myelocytes, eosinophil myelocytes, normoblasts, and megaloblasts, a differential count was not made. There are numerous mitoses, and fat is present in small amount.

Spleen.—The Malpighian bodies are inconspicuous and the pulp appears more uniform as a consequence, and resembles marrow. The cells are mainly large mononuclear cells. Some of these have very large pale nuclei, others have deeply staining irregular nuclei and show mitoses. There are a few eosinophil cells, small lymphocytes and erythrocytes.

Kidneys show inter-tubular deposits of large mononuclear cells with some lymphocytes.

Stomach shows its small vessels full of large mononuclear cells. There are mast cells in their neighborhood and in inter-tubular spaces of mucosa.

Lymph Glands.—The germ centers are composed of lymphocytes with some large mononuclear cells. The germ centers are inconspicuous in some areas. The medulla contains many large mononuclear cells with big pale, rounded, or again deeply staining irregular nuclei. Some cells are of great size. Numerous mitoses are present, especially in lymph sinuses suggesting endothelial proliferation. The lymph sinuses and also the capillaries are choked with large mononuclears, which have in places penetrated the capsule and passed into the perilymphatic fat.

Treatment.—The drugs used were arsenic and atoxyl.

Blood examination:

CASE I.—July 6, '07.

Red cells	2,922,800 per cmm.
White cells	7,200 per cmm.
Hæmoglobin	56-58%
Index	0.973

There are 21 per cent of polynuclears and 78 per cent of lymphocytes. Many of the lymphocytes are atypical, being large with much cytoplasm.

August 18, '07:

Red cells	1,888,000 per cmm.
White cells	3,200 per cmm.
Hæmoglobin	46%
Index	1.15
Polynuclears	46.0%
Eosinophiles	1.7%
Lymphocytes	46.7%
Transitionals	2.0%
Myelocytes	2.0%
Mast	0.9%

There were nine megaloblasts, thirteen normoblasts and four microblasts.

September 8, '07:

Red cells	3,360,000 per cmm.
White cells	4,000 per cmm.
Hæmoglobin	40.0%
Index	1.7
Polynuclears	35.8%
Small lymphocytes	60.0%
Large lymphocytes	3.7%
Transitionals	0.4%

Two megaloblasts, fifteen normoblasts and five microblasts.

September 18, '07:

Red cells	1,360.000 per cmm.
Leucocytes	6,900 per cmm.
Hæmoglobin	26.0%
Index	1.0%

There were 30.3 per cent polynuclears, 51.2 per cent small lymphocytes, 12.0 per cent large lymphocytes, 3.5 per cent transitionals, and 2 per cent myelocytes.

Remarks.—In 1906 I recorded a case of leukanæmia (myeloid splenic anæmia) at the Pathological Society of London. The patient was a male, aged 26 years, under the care of Dr. D. Hall, and had been jaundiced for years. There was great enlargement of the liver (59 oz., 1770 gr.) and spleen (42 oz., 1260 gr.). The blood had the features of pernicious anæmia as seen in a high index, low red cell count, and number of nucleated red cells; whereas the leucocytes were about normal in number but contained myelocytes and transitional cells in small numbers. The liver capillaries were stuffed with mononuclear cells, myelocytes, and nucleated red cells, and similar cells were found in the spleen.

Similar findings were made in the case now described, although lymphocytes in the blood and viscera were in excess of myelocytes. In both cases evidence of hæmolysis was absent.

Dr. Drysdale has collected and described 12 cases of this disease in the *Quarterly Journal of Medicine*, Vol. I, No. 1, October, 1907.

As to classification, Von Leube was led to place the disease under the name of leukanæmia on account of the combination of red and white blood cell changes found. Drysdale, on the other hand, considers that the blood picture of pernicious anæmia (which he believes to be always secondary or symptomatic) may be found in simple anæmia (post-hæmorrhagic) and in infections (bothriocephalus latus).

Drysdale, therefore, regards the anæmia in this disease as a severe secondary anæmia differing only in degree from the anæmia seen in ordinary leukæmia. Thus my cases would be regarded as atypical leukæmia, myeloid and lymphatic respectively, with a leukæmic or pseudo-leukæmic blood state. For my own part I attach more significance to the presence of nucleated red cells in these cases than does this observer. Thus, besides megalocytes there were 2200 megaloblasts and 370 normoblasts per cmm. in Drysdale's case, and 14,000 megaloblasts in Hurter's. This is not seen in the anæmia of sarcomatous disease nor usually in lymphatic leukæmia. In myelocythæmia when there is little or no anæmia and the patient is rosy and even vigorous, the presence of numbers of normoblasts has been frequently observed and also of microblasts and megaloblasts. There is then no *necessary* connection between these cells and anæmia. Therefore leukæmia or lympho- or myelocythæmia is, strictly speaking, not a term sufficiently comprehensive. Moreover the anæmia in these cases is of the pernicious type while the leukæmia is *per se* minimal, so that the use of the term leukanæmia is descriptive.

CASE II.—Lymphatic leukæmia with malignant lymphomata. A. V., aged 23 years; unmarried; domestic servant. Admitted under the care of Dr. E. Maynard, at the Sussex County Hospital, on September 11, '07, for "pleural effusion."

Past history of measles and anæmia, but none of phthisis.

Family history good.

Present History.—One month ago began to feel weak and to lose her appetite. She caught a chill, had a cough without expectoration, and hot flushes; she had no shivering attacks, no vomiting or diarrhœa; she had a feeling of "tightness" after food. On getting up in the morning her face, especially below the eyes, was very swollen. She passed urine without pain or

frequency. She went to a doctor a fortnight before admission because she was weak, but she had no pain in the chest; she gradually became short of breath, her cough was worse and she came to the hospital. She has had no headache or swelling of legs.

On Admission.—She was a pale, well-nourished woman, with with marked dyspnœa giving a history of two weeks' shortness of breath and cough, without pain.

Tongue was foul, with thick, white fur; teeth fair; breath offensive.

Chest.—Cardia, apex beat in fifth space two and one-half inches outside nipple line; left border, two and one-half inches outside nipple line; upper border, fourth rib; right border cannot be defined, owing to dullness of right chest; sounds clear, no murmur. Pulse 140, volume fair, tension normal; respiration 38.

Right Lung.—Percussion dull front and back; as high as clavicle in front and spine of scapula behind. Above spine note is hyperresonant. Vocal fremitus absent; vocal resonance diminished. Breath sounds very weak and scarcely heard over lower five inches.

Left Lung.—Resonant all over, no adventitious sounds heard.

Abdomen.—Liver: upper border cannot be delineated; left border, at costal margin, edge not felt; spleen, not felt.

Nervous System.—No abnormality.

September 12, '07. Right chest aspirated and 42 oz. of yellow serum were removed. Patient was then comfortable and of good color, but one hour after aspiration she became cyanosed and her dyspnœa increased. Ether and ammonia relieved her, and she brought up some frothy expectoration.

September 14, '07. Right lung gave a resonant note to the sixth rib in front. Breath sounds well heard, high pitched. Note behind resonant except for a hand's breadth at base which is dull. Over this area vocal fremitus was absent and vocal resonance diminished. Above this breath sounds were well heard, high pitched; there were numerous "bubbling" râles with inspiration in front, side, and back of chest. Breathing comfortable, slight expectoration, good color.

Blood Examination (see chart).

September 21, '07. 72 oz. clear yellow fluid removed from right chest, followed by collapse, which improved next day. X-rays applied to spleen five minutes daily.

September 27, '07. 76 oz. drawn off right chest.

October 9, '07. 32 oz. removed from right chest; this contained no tubercle bacilli, but excess of lymphocytes. The sputum was free from tubercle bacilli.

October 19, '07. Spleen: Lower edge level of anterior superior spine. Liver one and one-half inches below costal margin, very tender, smooth.

October 31, '07. X-rays applied to spleen for five minutes daily; smaller.

November 5, '07. Glands larger generally, not tender. The temperature varied from 97° to 103°, usually rising to 100° or 101° daily.

Post-Mortem Examination.

December 10, '07. Made by Mr. Wootton, H. P. (It was made outside the Sussex County Hospital and a brief report only was obtained.)

Body pale, but no loss of fat.

The liver is enlarged, fatty, but there is no new growth seen (weight three pounds, four ounces).

The kidneys show no abnormality (weight four and one-half ounces each).

The spleen is very large, firm, and no deposit growths are seen (weight two pounds).

The lungs show no new growth about the bronchi or elsewhere, but there is bloody pleural effusion.

The lymph glands are generally enlarged, both the superficial and deep; those above right clavicle are especially increased in size and also those in the right axilla and around the pancreas and spleen, where they are very dark in color. The mediastinal glands are the size of a cocoanut and appear to be malignant.

The ovaries and uterus appear normal.

The alimentary tube shows no affection of the lymphoid follicles of the walls or mucous membrane.

There were no hæmorrhages other than those into the lymphatic glands.

Microscopical Examination.

Liver (Lab. No. 2740).—The perilobular zones, the hepatic capillaries, and the capsule of Glisson show lymphocytes in numbers.

Marrow of ribs and sternum shows marked excess of lymphocytes with many nucleated red cells.

Cervical glands show lymphocytes in a reticulum with many foci of large round cells ("germ centers"). Lymphocytes are aggregated in the sinuses and capsular lymphatics. There are large areas of necrosis. The capsule is thickened and invaded by lymphocytes. Within the capsule of the gland which is probably not the normal gland capsule are "germ centers" evidently the site of new gland tissues.

There are many large cells with large pale staining nuclei whose cytoplasm is basophilic and not quite clear, which are of endothelial nature—some may be called giant cells.

Mediastinal glands show same characters as cervical, with higher development of the stroma. The lymph sinuses are choked with lymphocytes; and the lymphocytes are invading the capsule and the adjacent muscular tissue.

Peripancreatic glands like cervical. There are giant cells with single nuclei and hæmorrhages into the glands are present.

Ovaries show numerous round cells in stroma and in vessels, and around corpora albicantia and lutea.

Kidneys contain some foci of round cells.

Suprarenals.—Capillaries are stuffed with lymphocytes and there are masses in connective tissue of capsule.

Spleen is congested. Its vessels, capsule and framework are hyaline. There are many lymphocytes in sinuses.

Pancreas shows auto-digestion.

Blood examination:

Case II.—October 18, '07:

Red cells	4,056,000	per cmm.
White cells	222,200	per cmm.
Hæmoglobin	80.0%	
Index	1.0%	

Leucocytes:

Polynuclears	8.4%	18,664	per cmm.
Eosinophiles	0.5%	1,111	"
Small lymphocytes	65.1%	144,430	"
Large lymphocytes	17.4%	38,662	"
Transitionals	1.4%	3,110	"
Myelocytes	7.0%	15,554	"
Eosinophilic myelocytes	0.6%	1,332	"

Three monoblasts; megalo-, micro-, and poikilo-cytes present.

October 24, '07:

Red cells	3,328,000	per cmm.
White cells	124,000	per cmm.
Hæmoglobin	70.0%	
Index	1.0%	

Leucocytes:

Polynuclears	10.6%
Eosinophiles	0.7%
Small lymphocytes	50.6%
Large lymphocytes	26.7%
Transitionals	1.0%
Myelocytes	9.4%
Mast	0.3%

November 5, '07:

Red cells	3,350,000 per cmm.
Hæmoglobin	64.0%
Index	0.86%

November 14, '07:

Red cells	2,138,000 per cmm.
White cells	66,138 per cmm.
Hæmoglobin	50.0%
Index	1.1%
Polynuclears	8.4%
Small lymphocytes	85.0%
Large lymphocytes	3.6%
Myelocytes	3.6%

Remarks.—It is clear that there exists in this case a generalized disease of the lymphatic glandular system together with a leukæmic (lymphocytic) condition of the blood, liver, kidneys, spleen and bone marrow.

The question arises, of what nature was the glandular enlargement which I have labelled malignant lymphomata, and what is its relation to the blood condition (if any)?

As bearing on the character of the glandular enlargement, one may recall the following facts:

(1) The enlargement of the glands in leukæmia is usually within the limits of their capsules and contiguous glands do not fuse. Histologically the enlarged glands present a more or less normal appearance, the chief difference being the great increase in quantity of the germ center material which may equal or exceed in quantity that of the normal medullary substance. The reticular structures remain normal.

(2) Gibbons records 9 cases of Hodgkin's disease (lymphadenoma) with 5 autopsies in which the blood picture was normal and there was no relative change in leucocytes. The glands showed proliferation of the germ centers of follicles and of the endothelium of the sinuses, and the presence of giant cells, eosinophiles, a few plasma cells and polymorphonuclears. Gibbons also describes infiltration and penetration of the gland capsules with invasion or destruction of the adjacent muscle or other tissue. Metastases were present in the liver and spleen in 4 each, in the kidney in 2, in the lungs, pericardium and pancreas in one each. The metastases resembled gland tissue sharply defined but without a capsule and showed malignant invasive characters.

(3) Lymphosarcoma according to Kundrat, quoted by Salaman (*Trans. Path. Soc.*, Lond., 1904, "Sarcoma of Stomach") is an entity with the following characters. It is composed of lymphoid cells in a fibrillary meshwork and either cells or meshwork may be in excess, giving rise to medullary or fibroid forms.

It infiltrates "without restraint," thus differing from leukæmic and pseudo-leukæmic growths. Presumably this means that its infiltrating powers are more developed and destructive.

It chooses the readiest paths for propagation, does not show degenerative changes common to leukæmic and pseudo-leukæmic growths and its secondary deposits are not true metastases. It possesses other negative characters. It commonly begins in the glands of the neck or axilla.

(4) True sarcoma of the lymph glands presents the usual features of this growth elsewhere.

It will be seen that the glandular enlargement presents at once characters described in leukæmia, lymphosarcoma and, even in lymphadenoma.

Thus in some regions the enlargement of the lymph glands had not transgressed their capsular boundaries and there was a marked increase in germ center material with degenerative changes. In addition there was the blood and visceral condition which presented a typical leukæmic picture.

Again, in the mediastinum the growth presented certain of the characters of a lymphosarcoma in large areas and had fused into a large mass.

Lastly, the infiltration of the gland capsules and invasion of adjacent tissues is described in lymphadenoma, by Gibbons. In this disease, which is one of youth and early middle age, the commencement is usually in a gland or group of glands which gradually or rapidly enlarge.

Similarly Salaman had seen a case of lymphatic leukæmia where the glands had fused and formed a solid mediastinal mass which seemed to possess most of the malignant properties of lymphosarcoma. Also a lymphadenoma of the glands of the neck which gave rise to a large mediastinal tumor which had all the appearance of a lymphosarcoma and had invaded the pericardium and tissues at the base of the heart.

The relationship which I suppose to exist between the glandular disease and leukæmic blood condition I will give after the description of Case III.

CASE III.—Large round cell sarcoma of cervical glands of neck with changes in the leucocytes.

J. W. I., aged 39 years. Admitted under the care of Mr. Verrall, on November 21, '07, for swelling of the neck.

SUMMARY OF CLINICAL CONDITION.

There is a swelling of the lymph glands on the left side of the neck which started as a small lump nine weeks previous to admission. It gave rise to no inconvenience until two weeks before admission, when it began to be painful at night. There was no discomfort in eating or affection of the movements of jaws. There was "nagging" pain in left shoulder, arm and side of head.

An abscess, supposed to be septic, was opened at Guys Hospital, in 1905.

There was a large diffuse brawny swelling on the left side of the neck. There was no sign of metastases, no hæmorrhages. Microscopical examination of a small portion removed for diagnosis showed it to be due to a large round cell sarcoma of the lymph glands undergoing necrosis.

Trypsin and amylopsin were administered without obvious effect.

Blood Examination, December 14, '07:

Erythrocytes	5,500,000 per cmm.
Leucocytes	13,400 per cmm.
Hæmoglobin	108%

Leucocytes:

Polynuclears	69.0%
Eosinophiles	3.0%
Small lymphocytes	18.7%
Large lymphocytes	2.1%
Transitional cells	6.4%
Mast cells	0.4%

The blood shows 6.4 per cent of *transitional* cells. These were three or four times the size of red blood cells. The nuclei were rounded, but indented, and without granulations; or deeply indented and with slight, fine granulations.

Remarks.—The transitional cell is regarded as a large mononuclear in which the protoplasm has developed a fine scanty neutrophil granulation, the staining of the nucleus has improved, and the shape changed to a horse-shoe or hour-glass form.

This cell is rare in normal blood and difficult to recognize (Buckmaster). It is supposed that in the blood and elsewhere the large mononuclear cells change by becoming granular into transitionals and these again into the polymorphonuclear cells. There is no positive evidence of this, however, but Buckmaster has seen both large mononuclear cells and cells indistinguishable from transitionals in lymphoid tissue. There are appearances in the sections of this sarcoma which suggest this to be the case here. The granulation of the cell is thought to be a feature of the age of the cell.

Although Ehrlich considers that large mononuclear cells have no relationship with lymphocytes, it is thought by some that they are members of the lymphocyte series, and in this I agree.

Ehrlich considers that large mononuclear cells do not arise in the lymph glands, but Buckmaster has seen them in lymphatic glands and possibly they are macrophages. Possibly the bone marrow (Ehrlich), lymphatic glands (Gulland), and spleen (Turk) each contribute a small supply to the blood.

Summary.—(1) These cases afford evidence that the glandular enlargements and visceral deposits of leukæmia are closely allied to lymphosarcoma and that pseudo-leukæmia and lymphadenoma possess the characters of malignant growths.

(2) In leukæmia the lymphocytes may be regarded as elements of malignant character.

(3) In sarcomatosis, and sarcoma of the lymph glands changes may occur in the leucocytes (in the presence of transitional cells).

(4) No absolute distinction can be drawn between leukæmia and sarcoma.

THE TEACHING OF PHYSIOLOGICAL PATHOLOGY AT THE UNIVERSITY OF MICHIGAN.

By F. Peyton Rous, M. D.,
Instructor in Pathology, the University of Michigan.

During the last two years there has been offered to junior students at the University of Michigan an elective course in physiological pathology. For some time previous—indeed, since 1895—Dr. Warthin had taken up in the Journal Club themes in experimental pathology. This work he will himself report later.

The present course in physiological pathology extends over three hours a day during three weeks, and it is given twice in the year. No attempt has been made to take up the whole subject, but teaching has concerned itself mostly with those disturbances of circulation and respiration important clinically. No text-book is followed. Experiments and conferences on the topics illustrated by them have made up the major part of the work, but museum specimens have been shown when needed to bring out special points. Where possible the operative work has been accomplished previous to the meeting of the class, by the help of some of its members.

The course presented last autumn had the following arrangement:

I. Vascular Disturbances.

Anæmia, congestion, stasis, hæmorrhage, clotting, and embolism were studied in the mesentery of the frog. By ligation of the veins of the tongue the characteristic passive congestion described by Cohnheim was obtained. The capillaries and small veins became solidly packed with cells; they showed "mulberry protuberances" from which diapedesis took place; and in the arteries a pronounced *va-et-vient* rhythm became evident.

Ligation of the large blood-vessels in the dog showed the relative effects of occlusion of the femorals, the carotids, the cœliac axis and the portal vein. Ligature of the vessel last named brought the animal to a condition of intense dyspnœa and low arterial blood-pressure, from which it speedily recovered when the ligature was removed. In a dog suffering from shock some of the larger arteries were ligated to help recovery, thus following out the principle of a tight bandaging of the extremities, and of Crile's rubber suit.

The results on blood-pressure of handling the intestines, of repeated small hæmorrhage, and of one brisk hæmorrhage, were now tried. When loss of blood had brought its characteristic symptoms an infusion of salt solution into a vein was practiced. After the recovery of the animal this infusion was not discontinued but a bulk of fluid three times that of the animal's original content of blood was allowed to enter

the circulation—thus repeating the experiment of Cohnheim, and Lichtheim, and of Magnus. This caused marked dyspnœa which was not fatal. The arterial blood-pressure, once the animal had recovered from the effects of the hæmorrhage, was not altered by the additions of fluid. The pathological findings at autopsy after this procedure formed a good basis whereon to found a discussion of the causation of œdema. No anasarca, hydrothorax, or ascites was present, but only the well-known œdema of the stomach, gall-bladder, and especially of the pancreas, which was as though set in jelly. The effects of the infusion when the animal had been subjected to laparotomy and the intestines and mesentery had been handled gave an interesting side-light. In two such instances ascites developed. It was also noted that as infusion progressed the abdominal wound bled anew, oozing throughout its length. This observation has a direct bearing on the question of infusion after hæmorrhage.

Other results of vascular disturbance and of changes in the chemical constitution of the blood were shown by the effect on urinary secretion of ligation of a renal vein, of cutting one kidney out of the circulation, of contracting the vascular bed in the kidneys while raising the general arterial blood-pressure (adrenalin), of hydræmic plethora, and of increased sugar in the blood.

The demonstration of air and fat embolism will be mentioned farther on. An attempted observation of retrograde metastasis did not lend itself to a satisfactory graphic demonstration.

In all of these instances records were taken of blood-pressure in a common carotid artery and in an external jugular vein.

II. Cardiac Disturbances.

It was found possible to show in one afternoon and on one animal the effects of several lesions. Thus, pericarditis with effusion was simulated by introduction of oil into the pericardium, then an aortic stenosis was produced, and later a mitral insufficiency. Recent literature makes unnecessary a description of the means used to obtain these lesions. Tracings were taken from an auricle, a jugular vein, a carotid, and in the case of pericarditis of the intrapericardial pressure to demonstrate how small this may be while seriously interfering with the circulation.

Lesions of all of the heart-valves except the pulmonary were produced or simulated, though not all of these before the same class. In one instance the compensation in a case of mitral insufficiency was varied at will by the production of aortic stenosis. It was interesting to note the immediate rise of pressure within the left auricle and the greatly increased systolic pulsations which resulted from a very slight stenosis. In two cases of tricuspid insufficiency compensation was broken by the injection into a jugular vein of a little oil and a little air respectively. The air and fat embolism were slight, yet sufficient to overturn the balance of affairs.

A study of tricuspid insufficiency proved especially useful. It helped to demonstrate that, mechanically considered, the farther back in the heart-pump a lesion is, the more poorly it is compensated; and it brought out the influence of the time-element in heart-disease, for the venous stasis and pulsation, so characteristic clinically, do not at once follow tricuspid insufficiency abruptly produced in a dog. In one instance out of four, a systolic jugular pulsation slowly made its appearance during the minutes of observation.

While the class worked on these lesions its attention was incidentally directed to changes in the heart sounds. The thrill and murmur, perceived most strikingly at the left auricle, as a result of mitral insufficiency, demonstrated with a conclusiveness impossible in clinical teaching that the point on the chest wall at which we most easily feel a thrill or hear a murmur may correspond but ill with the cardiac region where these are most intense, and that such a point is valuable clinically only because it is on the line along which these signs are transmitted.

The damage wrought by a myocarditis was simulated in the usual manner by injection of a fixative into the musculature. In one instance these injections were confined to the left ventricle to note whether an acute pulmonary œdema would ensue, but the complication did not occur. An observation such as this, or such as the production of fat or air embolism, coronary occlusion, hæmorrhage, or asphyxia, was generally made upon an animal which had been used in a demonstration of a lesion not of itself fatal. The differences in mode of death between that consequent on sudden stand-still of the heart after coronary occlusion, on failure of the circulation after air embolism, despite violent cardiac contractions, on acute asphyxia, with its transiently heightened blood-pressure and persisting heart-beat, were brought out. In the right ventricle after air embolism the remarkable stiff, red foam was met with.

III. Disturbances of Respiration.

The effect on breathing of paralysis of the abdominal muscles of respiration and of the costal ones, respectively, was first observed. To simulate the paralysis a bandage strapped about the abdomen or chest was used—a poor measure, since, of course, it was impossible to avoid some effects from direct compression. The changes in breathing were recorded, as in the later experiments, by means of an air-transmission tambour connected directly with the trachea.

An acute dilatation of the stomach by its inflation with air (Kelling), through a canula tied into the œsophagus, resulted in a rise in intrathoracic pressure, marked dyspnœa, and a gradual fall in blood-pressure. The distention was relieved by puncture in the left hypochondrium. The immediate death of the animal by almost simultaneous cessation of heart-beat and respiration took place. Another interesting death occurred through acute pulmonary œdema, which came on during the production of an artificial hydrothorax. The lung had not been pierced.

By filling the peritoneal cavity rapidly with salt solution an ascites was simulated and its results in dyspnœa and changes

in blood-pressure were followed. Attention was drawn to the prominent physical signs of this lesion, as to those of the gastric distention, but they were not studied.

The mechanical conditions of a tracheal stenosis were produced at will by pressure on a large rubber tube united end to end with the trachea. During a stenosis, in both phases of respiration, the characteristic long-drawn breathing with active expiration was noted. Inspiratory stenosis gave a lengthening of the corresponding phase of respiration with a fall in the intrathoracic pressure, permanent with the duration of the lesion; expiratory stenosis gave opposite results. In all three cases the blood-pressure rose. By means of a bulbous sound the right primary bronchus was occluded. The records showed that this did not cause a change of the respiratory rhythm, or of the carotid blood-pressure. These phenomena were made the theme of a talk on the disturbances of respiratory rhythm and of intrathoracic pressure seen clinically.

The last demonstration dealt with the effects of increased intracranial pressure on blood-pressure and on respiration. It was easy to show that the general effects of increased intra- and extradural pressure are the same. Tracings were obtained of the rise of blood-pressure and slower heart-beat, of the pronounced slowing and final disappearance of respiratory movements.

Most of the experiments described were performed within a single course of three weeks, since, as has been indicated, one animal often sufficing for several of them. With the exception of the cardiac insufficiencies they were found useful for class purposes. In the production of the insufficiencies a lesion that brings the heart rapidly to a stop frequently cannot be avoided. Altogether, out of some fifty attempts at various demonstrations, six have been failures.

The value of the work in its present limited scope must be measured less by the amount of ground covered than by the point of view given the student for his study of disease at the bedside. Then he will not be able to "open the back of the clock," as in these lesions of animals. But that an experience with these lesions will render more concrete to him the problems in human pathological physiology, and will directly aid him in the solution of some of them, cannot be doubted. At present one must be satisfied with this as the principal result of a short course in pathological physiology. If the subject becomes fixed in the recognized medical curriculum it will be possible to teach with more system those deviations from the normal physiology with which the doctor should be acquainted.

THE TONSILLAR RING AND THE PARANASAL SINUSES AS PORTALS OF ENTRY OF INFECTIOUS DISEASES.[1]

By Sylvan Rosenheim, M. D., Baltimore.

The term "tonsillar ring" includes the faucial, lingual, and pharyngeal tonsils as well as numerous isolated and combined lymphoid follicles found in the oral-, naso-, and laryngo-pharynx.

The various views regarding the functions of the tonsils can be conveniently divided into two groups: first, the theories advanced before Stöhr's discovery of the emigration of the lymphocytes from the interior to the periphery of the tonsils; and second, those advanced after the discovery.

The earlier writers, mostly anatomists, considered the tonsils as functionless filling-in organs, supporting Kolliker's view.

Morell Mackenzie in his work mentions a theory very commonly held, of the relationship of the sexual organs to the tonsils, based on their hypertrophy, which is seen often at the age of puberty.

Some clinicians have considered the tonsils of importance in the digestive functions. Bosworth thought that they acted mechanically in moistening the bolus of food. Rossbach believed they produced a saccharine ferment. Fox had the idea that the faucial tonsils absorbed superfluous salivary secretion and Spicer assumed that the pharyngeal tonsils acted in a similar way for the tears and nasal secretion.

That the tonsils are blood-forming organs has been suggested from time to time. The discovery of the germinal centers, *i. e.*, places in the follicles in which active karyokinesis and cell division is taking place, first gave rise to this view. To substantiate it, experimenters have determined a decrease in the number of lymphocytes in the circulation after extirpation of the tonsils. Pluder has described a constant emigration of newly-formed cells into the efferent lymph channels but Goerke has pointed out how impossible it is to demonstrate this. He says (1907) that the tonsils as a part of the lymphatic apparatus may share in the blood-forming work but by no means play an important rôle. In a recent work (1904) Wood gives his revolutionary conclusions in regard to the functions of the tonsils. He considers them the principal leucocyte formers. They give rise to the lymphocytes, which in turn are transformed into the various other forms of leucocytes. They arise, he says, not from the germinal centers but directly from the tonsillar crypts.

It has been thought that the tonsils, like the adrenals and thyroid, might produce an internal secretion. The experiments along this line have given contradictory results. Masini injected an extract of tonsils into various animals and noted a rise of the blood pressure, whereas Scheirer, performing similar experiments, noted a fall.

In 1884 Stöhr described a phenomenon that occurs constantly in the tonsillar tissue, namely, a wandering of the

[1] Paper read at the Southern Section of the American Laryngological, Rhinological, and Otological Society in Baltimore, December, 1907.

lymphocytes from the interior of the tonsils to the periphery. They force themselves or are forced between the epithelial cells, injuring them and destroying their function. The open spaces thus made between the epithelial cells led Gerhard to consider the tonsils as physiological wounds. Some writers believe that the tonsils are rendered much more vulnerable by this action of the leucocytes, to deleterious substances and micro-organisms which might readily penetrate the tonsils, whereas others draw attention to the fact that the constant streaming out of the leucocytes might act as a protective agency. The two opposing views have been termed the protective and the infective theories.

To determine whether foreign bodies and micro-organisms penetrate the tonsils, Hodenpyl, Goodale, Hendelsohn, Lexer, Goerke and others have performed numerous experiments. Hodenpyl (1891) smeared human tonsils with olive oil and lard, and applied for from fifteen minutes to one-half hour, carmine and Berlin blue in a similar manner in dogs. He also used solutions of anilin colors and atropin. He concluded from his experiments that the tonsils absorbed neither fluid nor solids under ordinary circumstances. He thought that the rarefaction of the tonsillar epithelium explained the entrance of the toxin of diptheria into the general circulation.

Goodale (1898) and Hendelsohn (1899) arrived at different conclusions from their experiments. Goodale's experiments were performed on the human hypertrophied tonsils and seem most convincing. He injected aqueous carmine solution into the tonsillar crypts and then excised the tonsils at intervals of from twenty minutes to two hours. In two control cases he excised the tonsils immediately after injection and found no absorbtion of the coloring matter. In all the others he found that carmine particles had been absorbed and that the depth at which they were discovered varied directly with the interval of time which had elapsed since the injection. Hendelsohn arrived at similar results after simply blowing colored particles over the tonsils.

Goerke (1907) claims to have arrived at negative results in repeating these experiments and ascribes the positive results to two factors: first, to the mechanical force used in the experiments and secondly, to a reversal of the lymph stream due to the amount of powder employed. He thinks that bacteria penetrate more readily the mucous membrane of the pharynx. He mentions some experiments of Lexer in which virulent bacterial cultures were painted over the throats of animals and very few were found to penetrate the tonsils. He considers the lymphoid tissue of the throat to be the portal of entry. He mentions some cases reported by Menzer of acute articular rheumatism following angina in which bacteria were found deep in the pharyngeal mucuous membrane, and but few and these only superficially in the tonsils. He thinks the tonsils owe their protective function not only to the mechanical influence of the constant stream of leucocytes towards the surface but to two other factors, namely, the bactericidal action of the lymph, and that of the products set free by the breaking down of the lymphocytes. That infections occur through the tonsils, Goerke doesn't deny; that they do not occur more frequently is due to the protective function of these organs. He says the tonsils reach their greatest development in childhood, when their need is greatest, as the infectious diseases are then most common, and that they retrograde after this age.

Whatever may be the true function or functions of the tonsils, I am inclined to agree from my studies and the results of a careful reading of the literature, with Bosworth, that normal tonsils should not be seen. Goodale's experiments were done on hypertrophied tonsils and certainly showed that such diseased tonsils absorbed foreign materials. The evidence as collected from the literature is also overwhelmingly in favor of this view. As we shall see, many of the tonsils in severe septic infections appeared normal on the surface, whereas below the surface an entirely different picture was seen. It would not answer, however, to restrict the term diseased tonsils to those which project beyond the faucial pillars, as some of the most badly diseased tonsils are those which are hidden. The history of repeated attacks of sore throat sometimes attracts attention to them, or their morbid condition is discovered in searching for a focus of a so-called cryptogenic infection. I think that everyone is agreed that any pharyngeal lymphoid tissue that is seen should be removed. This tissue is subject to all the diseases that affect the faucial tonsils, and is probably much more frequently inflamed than is supposed, as owing to its position the inflammation is overlooked.

As acute or chronic arthritis was a common sequel to the cases of tonsillitis seen by me, I shall review briefly the literature of the other infectious diseases associated with diseases of the tonsils, before discussing the rheumatic cases.

Aneurysm.—Libman reports a case of mycotic aneurysm, which he ascribed to a tonsillitis which occurred three weeks previous to its occurence. Death resulted after operation and at autopsy streptococci and staphylococci were grown from the aneurysmal sac.

Appendicitis.—Cases have been described by Apolant and Kretz.

Erysipelas.—According to Gerhard focal erysipelas frequently has its origin in the naso-pharynx.

Meningitis.—Perigord narrates the history of a case of fatal meningitis, in which at autopsy an abscess was found in the pharyngeal tonsil.

Iritis.—A case in which this complication occurred is recorded by Dock.

Pleuritis and *Pericarditis.*—Numerous cases have been reported by Hanot, Heddaeus, Dock, Jessen, Richardieu, Lermoyez, Metzner and others. In a case of Hanot, streptococci were found in both the tonsillar and pleural exudates. In Metzner's case the tonsils at autopsy showed collections of pus in the interior with an intact surface. Staphylococci and streptococci were found here, as in the pus from the mediastinum, pleural cavity and endocardium.

Pneumonia.—In a young girl seen by Jessen, an angina was followed in twelve days by pneumonia, pericarditis and nephritis. Streptococci were found in the tonsils. A blood culture was negative.

Paraplegia and *strasbismus* have been observed to follow tonsillitis by Emil Mayer and *parotitis* by Rosenberg.

Nephritis.—Leyden, Jeppe, Morse and others have laid stress on this complication of tonsillitis. Leyden and Morse call atten-

tion to the fact that some of these cases may develop into the chronic forms of nephritis.

Osteomyelitis.—Kraske and Mayer have recorded cases.

Phlegmon of the lower extremities following follicular tonsillitis has been reported by Trumbull.

Oophoritis and *Orchitis.*—Joal reports six cases of this complication.

Septic Infection.—Fraenkel, Heubner and Bahrdt, Jessen, and Babes relate some interesting histories. Fraenkel describes a case of retro-pharyngeal phlegmon, which began in the tonsil, followed by purulent pericarditis and pleurisy. Micrococci were found in the exudate on the tonsils and elsewhere. In another case of membranous disease of the pharynx and larynx, ulcerative endocarditis followed. Diplococci and streptococci were found. Heubner and Bahrdt saw a case of fatal scarlatina and acute articular rheumatism following membranous tonsillitis. The autopsy demonstrated a purulent exudate in the tonsil which had penetrated the capsule causing an infectious thrombo-phlebitis of the common jugular vein. Cocci in chains were found in the exudate on the tonsils, in the joints, blood, etc. Jessen reports two interesting cases. In one a woman was brought into the hospital comatose. There were hemorrhages into the skin. Albumin and casts were found in the urine. The tonsils and mouth were normal. Death occurred in twelve hours. Autopsy revealed abscesses in the interior of the tonsil on section, the outer surface appearing normal. The spleen was large and soft. Numerous abscesses and hemorrhages were found in the kidneys. In the second case the disease began as a follicular tonsillitis which was followed by double pneumonia, pericarditis and sepsis. On sectioning the tonsils abscesses were found, the surface being intact. Staphylococci were found in the organs.

Skin Manifestations.—Erythema, erythema nodosum, purpura, and erythema exudativum multiforme have been described by Jessen, Stephanides, Joal, Finger, and others. In a case seen by Jessen, erythema nodosum and pains in the joints followed three days after the onset of an acute follicular tonsillitis. Streptococci and staphylococci were found in the tonsillar exudate in pure culture. A blood culture was negative. In the case of a woman subject to attacks of purpura and polyarthritis seen by Stephanides, removal of the tonsils was followed by recovery. Finger found streptococci in the skin of a patient with erythema exudativum multiforme.

Typhoid beginning with an angina has been noted five times by Capalarri.

Tuberculosis.—The frequency of tuberculosis of the tonsillar ring is now so generally recognized that it is rare to come across an author like R. C. Brown, who declares this to be infrequent, and thus argues in favor of the protective function of the tonsils. Mayer originally expressed the idea in a vague way that enlargement of the lymphoid tissues in the throat might in some way be related to scrofula. Lartigau and Nicoll in an article written in 1904 have given an extensive review of the subject, together with the result of their examinations of seventy-five pharyngeal tonsils. Parts of each tonsil were used for inoculation experiments, the rest were examined histologically. They found that 16% were tuberculous and 10% showed histological lesions of tuberculosis. The lesions are usually close to the surface and focal in character. They considered that the tonsils might be portals of entry for tuberculosis. From a careful histological examination of sixty-four adenoids Uffendorfe found that 4-5% were tuberculous. Two of the patients gave no tuberculin reaction after the removal of the adenoids; one patient who, however, had pulmonary tuberculosis, reacted to tuberculin, after extirpation of the adenoids. The author concluded from his studies that the lesions were purely focal. Moritz Schmidt says it is remarkable how infrequent tuberculosis of the tonsils is when it is seen so often post mortem (Strassman 8%, Dmochowsky 39%, Schlenker 8%). However, the later researches, as those of Lartigau and Nicoll, have shown a high percentage of tuberculosis in adenoids removed during life. Often numerous specimens have to be examined before this is demonstrated. The *B. tuberculosis* penetrates the tonsillar epithelium in the lower animals without leaving any traces (Theobald Smith) and there is no reason why the same should not occur similarly in human diseased tonsils. In a number of cases of cervical adenitis in which the tonsils on removal were carefully examined, both histologically and by inoculation of animals, Goodale found a large number to be tuberculous. The prompt clearing up of the glandular condition in these cases demonstrated the primary nature of the lesion in the tonsils.

The literature showing the relationship between the various forms of rheumatism and tonsillitis is very extensive. The connection between acute articular rheumatism and angina has been noted for a long while. Heberden in 1804 described an angina which terminated in acute articular rheumatism and Trousseau in 1865 revived this idea. Friedrich points out that it was one of the first diseases in which the tonsils were recognized as a portal of entry for infectious diseases. The importance of angina as an etiological factor was also early pointed out by Lagranière, Boeck, Loebl, Mantle, Lennox Brown, Fowler, Cheadle, and others. The literature of the subject is completely covered in the articles of Buss, Suchannek, Bloch, and Peltersohn.

St. Clair Thompson says that 30% of the cases of acute articular rheumatism are preceded by angina. According to Ingals 13-29% of cases of acute articular rheumatism and tonsillitis have an identical cause.

Stoeffel, Stephanides, and Gurich have reported cases of cures of the arthritis following proper treatment of the tonsils and the latter makes this a point in favor of the tonsil being the portal of entry.

A great variety of organisms, staphylococci, streptococci, pneumococci, etc. have been found in angina. Likewise various organisms have been found in acute articular rheumatism. Poynton and Paine in eight cases found a diplococcus which they demonstrated in the tonsils, the pericardium, the heart valves, and the rheumatic nodule. They review the literature and state that the following organisms have been obtained: staphylococci and streptococci, diplococci and anærobic bacilli resembling anthrax.

I shall describe first briefly the technique and findings in a small series of cases in which I removed the tonsils, which were studied bacteriologically by Dr. J. C. Meakins. Most of these were from the wards of the Johns Hopkins Hospital and represent the cases of acute articular rheumatism admitted during a short period. These cases seem to me to be of importance and interest, as they furnish additional evidence to prove that the tonsils, and tonsillar ring are frequent portals of entry and foci of infection. All were adults whose faucial tonsils were diseased. The tonsils were enucleated under cocaine anæsthesia by Worthington's method. They were carried in sterile receptacles to the laboratory and cultures taken in three ways. These Dr. Meakins classifies as external, internal, and autolized cultures. The external were taken from the depths of the crypts; the internal through a

sterilized cut surface from the interior and grown on slant glycerine-agar and agar plates. The autolized were made as follows: the tonsils were first washed for one minute in a 1-1000 solution of bichloride of mercury, then in sterile salt solution; after this they were placed in either broth- or milk-culture flasks.

External Cultures.—In all of these streptococci and staphylococci predominated. Other organisms, bacilli or diplococci, were also found.

Internal Cultures.—In five cases the streptococcus was found in pure culture; in four streptococci and a few staphylococci; in one streptococci and a few bacilli, and in one no organisms.

Autolized Cultures.—In seven cases a pure culture of streptococcus was separated. In one streptococci and a few other cocci (4 foreign colonies in 2 plates) were found; in one the *Staphylococcus pyogenes aureus* principally. In two the results were negative.

CASE 1.—Mrs. ——, æt. 40 years, white. Admitted March 18, 1907.

Diagnosis.—Acute articular rheumatism.

Complaint.—Sore throat and rheumatism.

Family History.—Negative.

Personal History.—Usual diseases of childhood, but no diphtheria or scarlatina. Has suffered from tonsilitis from her 13th year. When 14 years old had an attack of acute articular rheumatism affecting both ankles and wrists.

Present illness began 7 weeks ago with an attack of quinsy in which the abscess ruptured into the throat. Two weeks ago both shoulders, hips, knees, and elbows became very tender, painful, hot, and slightly swollen.

Physical Examination.—Joints above mentioned painful and tender but not swollen.

Slight mitral systolic murmur.

Very little tonsillar tissue seen. Slight amount of exudate in the crypts. Cultures from the throat gave an almost pure culture of streptococcus. The opsonic index to streptococcus was 0.65.

The tonsils on dissection proved to be much larger than expected, and belonged to the almost completely submerged variety.

May 1. Patient left hospital much improved, still having some pain at times and slight stiffness.

The internal and autolized cultures gave a pure growth of streptococcus.

Discharged May 1.

CASE 2.—Mr. ——, æt. 19 years, white. Admitted March 20, 1907.

Diagnosis.—Acute tonsillitis. Acute articular rheumatism. Acute mitral endocarditis. Ascaris and trichiuris infection.

Family and *Personal History.*—Unobtainable.

Physical Examination.—Joints affected. Pain in right elbow, hips, right knee, and behind neck; the elbow is hot, red, and swollen.

The tonsils on admission were large, red, and acutely inflamed. No exudate present. On the post pharyngeal wall is a thick greenish purulent secretion. Pus is seen in the right nares, about the middle turbinate and on the inferior turbinate. On exploration of the antrum about a week later a slight mucoid discharge was found.

April 12. Tonsils dissected out.

April 26. Still has at times slight pains in joints. Has improved very much and has gained weight.

Internal cultures showed pure streptococcus. Autolized cultures negative.

Discharged.

CASE 3.—Miss. ——, æt. 32, white. Admitted February 28, 1907.

Diagnosis.—Arthritis deformans. Acidosis.

Complaint.—Stiff and swollen joints.

Family History.—Negative.

Personal History.—No history of tonsillitis. Has had an occasional sore throat; as a child had "catarrh."

Present Illness.—Duration 4 years. Knuckles gradually enlarged, no pain. One year later the wrists were affected; they were swollen but not painful. Two years ago the shoulders were attacked. Now the right knee is swollen and painful.

Physical Examination.—Nasal examination revealed no signs of accessory sinus disease. A few diseased crypts were found at the apex of an atrophied tonsil. A slight amount of tonsillar tissue was removed by dissection. Cultures from this, internal and autolized, demonstrated streptococci alone.

She was treated with injections of dead streptococci, and when she left the hospital June 10 she was much improved.

CASE 4.—History lost.

CASE 5.—Mr. ——, æt. 29, white. Admitted March 27, 1907.

Diagnosis.—Acute articular rheumatism (2d. admission).

Family History.—Unimportant.

Personal History.—Negative. No history of tonsillitis or sore throat.

Present Illness.—In 1903 pain in various joints, but no redness or swelling. Was unable to walk or move legs.

Complains of pain in left knee and sterno-clavicular joints. Was hoarse and had sore throat several days before this attack.

Physical Examination.—Left knee joint swollen, hot and red. There is diffuse tenderness, and the right ankle is swollen.

A little muco-pus seen in left nostril about middle turbinate. Tonsils enlarged, otherwise not diseased.

April 19. Small fibrous tonsils dissected out. No rise of temperature following operation.

Internal cultures from tonsils showed streptococci and a few staphylococci. Autolized cultures contained only streptococci.

Discharged well April 30.

CASE 6.—History lost.

CASE 7.—Dr. ——. Tonsils removed as a prophylactic measure after an attack of tonsillitis. Patient was subject to frequent attacks of tonsillitis. These, however, had not been followed by any sequelæ. Both internal and autolized cultures showed only streptococci.

CASE 8.—Mr. ——, æt. 26, white. Admitted April 26, 1907.

Diagnosis.—Hypertrophied tonsils. Acute arthritis (convalescence). Mitral stenosis.

Complaint.—Patient is convalescent from an attack of acute arthritis and comes into the hospital to have tonsils removed.

Family History.—Unimportant.

Personal History.—Has had measles, mumps, pertussis, scarlatina, and rheumatism. Had scarlatina when 14 and rheumatism (knee joints swollen and painful) when 16 years old.

Present Illness.—Commenced March 8 with headache and slight sore throat; on March 13 the patient suffered from severe cough and later expectorated blood. On March 15, the knee joints became stiff and painful. These cleared up in a few days, and the patient began to have shortness of breath, and pain in the chest, running down the arm.

April 26. Large submerged tonsils were dissected out. Considerable hemorrhage at night necessitated the use of Monsel's solution. Temperature for two following days rose to 100.5° and

patient had slight earache. Both internal and autolized cultures showed pure growths of streptococci.

Discharged April 30.

Case 9.—Miss ——, æt. 32, white. Admitted May 20, 1907.

Diagnosis.—Acute infectious arthritis. Chronic tonsillitis.

Complained of enlarged tonsils.

Family History.—Unimportant.

Personal History.—Tonsils have been enlarged since childhood. No history of attacks of tonsillitis or quinsy.

Present Illness.—Five weeks ago had swelling of right hand and forearm, which was pronounced inflammatory rheumatism by her doctor. Had pains in various parts of her body. This lasted 2 weeks.

Physical Examination.—Knuckles of first and second fingers are swollen. Hands are held flexed and cannot be completely extended. Exostosis on right wrist. Both ankles slightly enlarged, motion is not impaired. Tonsils on dissection proved to be very badly diseased. The crypts were full of a cheesy-looking material. Temperature rose to 100° one day after operation, then fell to normal.

Internal cultures of these tonsils demonstrated streptococci and a few bacilli. Autolized culture showed pure growth of streptococci.

Discharged May 21.

Case 10.—Mr. ——, æt. 30 years, white. May 20, 1907.

Diagnosis.—Acute articular rheumatism. Hypertrophied tonsils.

Complaint.—Rheumatism.

Family History.—Mother suffers at times from rheumatism; otherwise unimportant.

Personal History.—Has had measles, diptheria, scarlatina, mumps, chicken pox, and typhoid fever.

Present Illness.—In the latter part of February had an attack of tonsillitis. This was immediately followed by pain in the left knee and afterwards in numerous joints. Has just come from Atlantic City where he has been during this illness.

Physical Examination.—The joints of the lower extremities are somewhat stiffened, though the function is fair. Slight thickening of the joints of the upper extremity.

Tonsils hypertrophied.

May 20. Tonsils were dissected out; they were much larger than they appeared to be. No rise in temperature after the operation. Urine negative.

December 12. Patient is in very good health. No signs of his rheumatic trouble (report from mother).

Internal cultures demonstrated mostly streptococci and a few staphylococci. The autolized cultures were negative.

Case 11.—Mr. ——, æt. 27 years, white. Admitted April 27, 1907.

Diagnosis.—Acute articular rheumatism. Chronic tonsillitis.

Family History.—Unimportant.

Personal History.—No history of attacks of tonsilitis. Had an acute coryza two weeks previous to this illness.

Present Illness.—Began three weeks ago with pain and swelling in the left ankle. Then the other joints of upper and lower extremity became affected. On admission the right elbow was acutely inflamed, and both shoulder joints and left ankle were very tender.

Physical Examination.—Joints affected as above described. Heart normal. Both tonsils very large. Pus seen in right nares, about the middle turbinate and over the inferior turbinate. Pharynx and post nasal space atrophic. Vocal cords reddened.

Tonsils removed May 23. May 31 a note made saying that arthritis has wonderfully improved.

July 2. Is now able to walk. Temperature at times goes up to 100°. No pain on passive motions of the joints excepting in the left knee.

Internal cultures of these tonsils were negative. Autolized cultures showed streptococci with four colonies of a foreign coccus in two plates.

Resume of Cases and Conclusions Regarding Tonsils.

In two of the series of cases which Dr. Meakins studied bacteriologically the histories have been lost. In one case the tonsils were removed as a prophylactic measure several weeks after an attack of tonsillitis. These were of the submerged variety and both internal and autolized cultures demonstrated streptococci in pure culture. Seven of the remaining eight were cases of acute articular rheumatism. In five of these, the tonsils were definitely hypertrophied, in two they were not. In four of them a streptococcus was found in pure culture, in two streptococci and staphylococci, in one streptococci and other cocci, in one streptococci and a few bacilli.

In one case of arthritis deformans streptococci were found in pure culture. This case was treated with streptococcus vaccine and improved markedly.

In five of the arthritis cases no note was made in the history of previous attacks of tonsillitis. At the onset of the disease in one case, the patient had "sore throat," another had quinsy, and another simple tonsillitis. One patient had two weeks previously an acute coryza. No note as to throat symptoms is found in the other histories.

Two of the cases had a serious complication, an endocarditis of the mitral valves. Both of these were cases in which the streptococcus was found in pure culture internally. In one of these the opsonic index to the streptococcus was 0.65. In another case of acute articular rheumatism complicated by mitral and aortic disease, streptococci were found in pure culture in the tonsillar crypts and in this case the opsonic index was 0.55.

All of these cases left the hospital a short time after the removal of the tonsils either well or much improved.

Besides this series of cases of acute arthritis, I have had a number of the more or less chronic variety, but sufficient time has not yet elapsed to draw conclusions. In all, however, the tonsils even though not apparently morbid, have disclosed evidence of disease after their removal. In some pus was seen to exude from between the anterior pillar and the tonsil, in others cheesy matter was expressed from the crypts. Even though this may not be seen on removing them, careful bacteriological examination will often reveal virulent organisms in the depths.

Our conclusions from the above observations are:

(1) That bacteria penetrate deeply into the substance of diseased tonsils.

(2) That all tonsillar tissue that appears hypertrophied should be treated or removed. All evidence points to the ready penetration of bacteria and injurious substances into their interiors, in many cases leaving no visible sign of such penetration. In the present state of our knowledge we have no method of pointing out the dangerous varieties of tonsils.

Any functions that they may perform, will be carried on by the tonsillar tissue that always remains even after the most careful removal. There are no reports in the literature of damage done by such removal.

(3) In all cases where there has been a history of repeated attacks of tonsillitis, this should constitute a cause for their removal, although they do not look diseased.

(4) In all infectious diseases the lymphoid tissue of the throat should be carefully examined as a possible focus of infection.

(5) The marked and immediate improvement in the series of cases reported, substantiates the above conclusions.

(6) The organisms found in the tonsils in the cases reported are probably the causal agents of the attacks of acute articular rheumatism.

THE PARANASAL SINUSES AS PORTALS OF ENTRY.

We are just as much in the dark in regard to the functions of these cavities as we are regarding those of the tonsillar ring. Accumulating evidence seems to point to them as a frequent focus of distant infections.

We have a number of carefully worked out series of autopsies on these cavities by various authorities. Fraenkel found 43.1% of diseased sinuses, Gradenigo 22% (antra only examined), Lapalle 32%, Martin 48%, Kirkland 32%, Wertheim 47%, Oppikofer 18%.

All authors agree in the great percentage of diseased cavities, but there is considerable difference of opinion regarding its significance. Fraenkel, Wertheim, Kirkland, Darling consider that the diseased cavities may be a source of general infection. Wertheim mentions three ways in which this occurs: (1) Swallowing the secretion; (2) aspiration of the secretion into the lower air passages; (3) absorption of toxins. I would like to add to this a fourth, namely, absorption by the blood or lymph of the causal organism. We must rely mainly on our clinical data to prove this, for as yet there are no more exact methods.

Kirkland examined the various cranial sinuses in 100 consecutive autopsies at the Sydney Hospital. He found a remarkably high percentage of the patients dying of pneumonia had suppurative disease of these cavities (50%); and believes that usually the sinus disease is secondary to the pneumonia; but he reports some interesting cases of pneumonia and peritonitis, bronchiectasis, bronchitis and gangrene of the lungs in which he considered the sinus trouble to be the primary cause. In two cases bronchitis were cured after treatment of the antrum of Highmore.

In 360 nasal obductions Wertheim found 165 cases of diseased sinuses, *i. e.*, 46%. Of these 96, or 23%, were cases of empyema. He also found a high percentage of pneumonia cases had sinusitis, 9 out of 16 cases, or 56%. He says that tuberculosis predisposes to sinus trouble, for he found 31 empyemata in 106 cases of tuberculosis, or 29%. Four cases of syphilis had empyemata. The author notes the difficulty in determining whether the disease is acute or chronic but thinks that the majority of his cases were of the chronic variety. He mentions the fact that even clinically in both acute and chronic sinus cases, on opening the sinus at operation secretion may be present in but small amount, or altogether absent. The usual findings in his cases were an infiltration of the epithelium with numerous polymorphonuclear leucocytes, round cell infiltration in the sub-epithelial zone, less in the periosteal zone, and an edematous infiltration of the entire tissue. A sclerotic condition was rarely found.

In a recent communication, Darling gives the results of 52 autopsies in which the various sinuses were examined. Of these 37 were cases of pneumococcus infections, 15 were of control cases. Of the pneumococcus infections 92% had purulent sinusitis, of the controls (anemia, intestinal trouble, broncho-pneumonia, etc.) but 28% had a sinusitis. Of the pneumococcus cases there were 22 cases of lobar pneumonia, 9 of acute meningitis, 1 of acute pericarditis, and 5 of septicæmia. This writer considered in all these cases of pneumococcus infection, that the changes in the sinuses indicated a chronic inflammation. He believes the accessory nasal sinuses to be the portals of entry of the pneumococcus.

In a recent article (1906), Oppikofer gives his conclusions on this subject. These are based on the results of 200 nasal obductions. He found 47% of the cases had inflammation of the sinuses, and of these about 25% were purulent. He says it is practically impossible to determine by histological examination whether these cases are acute or chronic, and considers that most of them are of the nature of terminal infections. In only 3 out of 23 cases did careful rhinoscopic examination show sinus disease.

In the case that I here report the relationship between the sinusitis and the joint trouble can hardly be disputed. It illustrates very clearly that it is necessary to examine these cavities as a possible focus of infection. Diagnostic methods are still imperfect and there is no doubt that even the most expert rhinologist overlooks cases of this kind. The great disproportion between autopsy statistics and clinical facts points to this conclusion. I do not agree with Oppikofer that the majority of these cases are terminal infections, first because in certain diseases (*e. g.*, pneumonia), such a large number of cases occur, and secondly the histological changes oppose this view.

Mr. ——, white, æt. 31 years. Carpenter.

***Diagnosis.*—Spondylitis. Right chronic sphenoiditis and ethmoiditis. The patient was referred to me by Dr. Baer for examination of the nose and throat as he could not lie on his back on account of secretion flowing into the throat.**

***Family History.*—Unimportant.**

***Personal History.*—Some years ago he had suppuration in the right middle ear, and has had nasal trouble for years. In July, 1906, he was treated in the Johns Hopkins Hospital for pain in the hips and in the back of the right leg. After a short rest in bed, he left the hospital much improved. In November, 1906, the pains returned. There was marked sciatica and the spine was stiff and bent to the right. The patient was scarcely able to sleep at night.**

***Present Illness.*—In January, 1907, he was again admitted to the hospital with the same condition. He was unable to bend the**

spine. He has had a discharge from the nose for six years. He has pain in the back of his head and his ears at times.

Physical Examination.—An abundant purulent discharge is seen in the right nares between the middle turbinate and the septum. The turbinates are atrophic. The left nares is negative. The pharynx and post nasal space are atrophic. The posterior wall is covered with muco-pus. The larynx is normal.

Treatment.—Up to this time the treatment of the spondylitis had been ineffective. He was unable to lie quietly on his back and the bakings did not do much good.

The right middle turbinate was removed and the posterior ethmoidal cells and sphenoidal sinus were laid wide open by means of Hajek's hooks and Cordes' sphenoidal instrument. There was considerable necrosis of these cells and they were thoroughly curetted. This operation was done in January. The improvement in the spondylitis was immediate. Dr. Baer said it cleared up as if by magic. In February the nose looked very clean. In March he was shown at the Johns Hopkins Medical Society, when his back was straight and there was no limitation of motion.

BIBLIOGRAPHY.

Adler, Isaac: Remarks on some general infections through the tonsils. N. York M. J., 1906, March, p. 641.

Beattie, J. M.: A contribution to the bacteriology of rheumatic fever. Brit. M. J., 1906, II, p. 1781.

Bloch, E.: Zur Aetiologie des Rheumatismus. Münch. med. Wchnschr., 1898, p. 445, and p. 488.

Brown, Lennox: Rheumatism and tonsillitis. Lancet, 1900, No. 2, p. 970. The Throat and Nose and Their Diseases, Lond., 1899.

Brown, R. C.: The relation of the tonsil to infection and infectious diseases. Med. Rec., 1907, p. 341.

Buss: Ueber die Beziehungen zwischen Angina und acutem Gelenkrheumatismus. Deutsches Archiv. f. klin. Med., Vol. 54, 1895, 1.

Darling, S. T.: The accessory nasal sinuses and pneumococcus infection. J. Am. M. Ass., Vol. XLVII, p. 1561.

Fraenkel, A.: Ueber septische Infection im Gefolge von Erkrankung der Rachenorgane. Ztschr. f. klin. Med., Vol. XIII, 1887, p. 14.

Fraenkel, E.: Beiträge zur Pathologie und Aetiologie der Nasennebenhöhlenerkrankungen. Virchow's Archiv, Vol. 143, 42.

Goerke, M.: Beiträge zur Pathologie der Tonsillen. Archiv f. Laryng. u. Rhin., Vol. 19, Pt. 2, 1907, p. 244.

Goodale, J. L.: Ueber die Absorption von Fremdkörpern durch die Gaumentonsillen, etc. Archiv f. Laryng. u. Rhin, Vol. 7, 190. The examination of the throat in chronic systemic infections. Transactions of the Am. Laryng. Ass., 1906 and 1907.

Gulland, G. L.: On the functions of the tonsils. Edinb. M. J., 1891, p. 435.

Heubner und Bahrdt: Zur Kenntniss der Gelenkeiterungen bei Scharlach. Berl. klin. Wchnschr., No. 44, 1884, p. 697.

Heymann: Handbuch der Laryngologie u. Rhinologie, Vol. 2, p. 496. Die Krankheiten der Rachentonsille. J. Gottstein u. R. Keyser.

Hodenpyl, E.: The anatomy of the faucial tonsils, etc. Am. J. M. Sc., 1891, p. 257.

Ingals, E. F.: The relation of tonsillitis to rheumatism. The Laryngoscope, 1907, p. 713.

Jacobi, A.: The tonsil as a portal of microbic and toxic invasion. Arch. Pediat., July, 1906, p. 481.

Jessen, F.: Ueber die Tonsillen als Eingangspforte für schwere Allgemeininfectionen. Münch. med. Wchnschr., No. 45, 1898, p. 709.

Kirkland, T. S.: The pathological conditions of the cranial sinuses. J. Laryng., 1902, p. 561.

Lartigau, A. J. and M. Nicoll: A study of hyperplasia of the pharyngeal lymphoid tissue (adenoids) with especial reference to primary tuberculosis. Am. J. M. Sc., 1902, p. 1031.

Lexer, E.: Die Schleimhaut des Rachens als Eingangspforte pyogener Infectionen. Archiv f. klin. Chir., Vol. 54, 1897, p. 736.

Libman, E.: Cases of mycotic aneurysms. Special reprint.

Mackenzie, Morell: Diseases of the throat and nose, Vol. I, p. 46.

Masini, G.: Abstract in N. York M. J., 1898, p. 419.

Menzer: Ueber Angina, Gelenkrheumatismus, Erythema nodosum und Pneumonia, nebst Bemerkungen, über die Aetiologie von Infections-Krankheiten. Berl. klin. Wchnschr., 1902, pp. 10 and 32.

Michelsen, M.: Ueber die etiologischen Beziehungen der akuten Entzündungen der Tonsillen zu anderen Erkrankungen, etc. Inaug. Dissert., 1901.

Oppikofer, E.: Beiträge zur normalen und pathologischen Anatomie der Nase und ihrer Nebenhöhlen. Archiv. f. Laryng. u. Rhin., Vol. 19, pt. 1.

Pirera: Centralbl. f. Laryng., Vol. XVI.

Poynton, F. J. and Paine, A.: The etiology of rheumatic fever. Lancet, 1900.

Saalfeld, M.: Ueber die sogenannte Pharyngitis Granulosa. Virchow's Archiv, Vol. 82.

Schmidt, M.: Die Krankheiten der oberen Luftwege, Berlin, 1903.

Spicer, S.: The tonsils. Their functions, etc. Lancet, 1888, Vol. 2, p. 805.

Stöhr, P.: Ueber Mandeln und Balgdrüsen. Virchow's Archiv, Vol. 97, p. 210.

Uffenorfe, W.: Beiträge zur Histologie der hyperplastischen Rachentonsille, etc. Inaug. Dissert, 1903.

Wertheim, E.: Beiträge zur Pathologie und Klinik der Erkrankungen der Nasennebenhöhlen. Archiv. f. Laryng. u. Rhin., Vol. 11, 901, p. 169.

Winslow, J. R.: The tonsil as a menace to the organism. Maryland M. J., 1899, p. 302.

Wood, G. B.: The functions of the tonsils. Univ. Pa. M. Bul., 1904, Vol. XVII, p. 246

Worthington, T. Chew: A Simple Method of Excising the Faucial Tonsil. J. Am. M. Ass., May 25, 1907.

NOTES ON NEW BOOKS.

The Diseases of Children. A Work for the Practicing Physician. Edited by Dr. M. Pfaundler and Dr. A. Schlossmann. English translation edited by H. L. N. Shaw, M. D. and L. La Fétra, M. D. With an introduction by L. E. Holt, M. D. In four volumes. (*Philadelphia and London: J. B. Lippincott Company, 1908.*)

After a rather commonplace introduction by Holt, we find the scope of the work outlined in the preface. It was the aim of the editors to secure the collaboration of investigators who had enriched the subjects treated by original contributions from their pens, and when we cast our eye over the names of the different authors it appears that they have succeeded admirably in most instances. The editors evidently recognize the modern tendency of scientific medicine to put the physiological aspect of diseases in the foreground.

The scope of the work renders it difficult and, indeed, impracticable to give it a searching review, and we must rest satisfied to mention the main chapters, adding a few remarks here and there.

The first chapter "General Pathogenesis and Pathology of Childhood" is written by Hamburger in his usual interesting manner.

In the "Symptomatology of Children's Diseases" Pfaundler has set himself the laborious and difficult task of tabulating the symptoms of children's diseases and pointing out their possible significance. The result may serve to tide the practitioner over many a lapse of memory and become a guide to his diagnosis. The author, however, knows the limited value of his undertaking. "Even the simplest mental picture of a disease can no more be resolved into the sum of several symptoms than can a portrait into the successive strokes of the artist's brush. The result in either case would be a caricature." Furthermore, the author does not claim a complete fulfillment of his task, nevertheless his labors will, no doubt, be rewarded by the assistance they will afford to many practitioners, even if there is still room for some improvement. The special index to this part is a welcome addition.

"General Prophylaxis in Diseases of Children" follows. Here Bendix touches—in parts more detailed—on the most important precautions to be taken in order to secure the child's bodily and mental welfare. The treatment of the subject is in some places too indefinite, particularly with regard to overwork—sometimes perhaps the opposite—in school and home studies.

Neumann, in the chapter on "General Therapeutics in Diseases of Children" expresses the opinion that loss of appetite, due to constipation, a frequent enemy to health, is most readily removed. If he has in mind those cases of chronic constipation, which are the bane of many physicians, we should be much indebted to him if he would kindly tell us how. His advice to pay a little more attention to the taste of the little patient suffering from loss of appetite may sometimes be heeded with advantage, but when he states "sometimes severe disciplinary measures lead to a quicker and better result than can be obtained by other means" he, no doubt, comes very near the truth; at the same time the majority of the parents in this country can hardly be persuaded to adopt any severe disciplinary measures under such circumstances. The paragraph on "Psychotherapy" is rather short, although the author recognizes its value fully—and, indeed, this therapeutic agent has received rather scanty notice in the treatment of children thus far. It may well be that hypnosis will find a somewhat wider range of application than the author is willing to concede to it. The paragraph on "Hydrotherapy" will help, we hope, to introduce this form of treatment more among our pediatrists. It is truly remarkable how thoroughly this most convenient and effective therapeutic measure has been neglected. The paragraphs on "Mechanotherapy" and on "Combined Physical Therapy" are well worth reading. The subject "Electrical Treatment" is dismissed with a few words.

Praussnitz, writing on "Mortality and Morbidity in Infancy" commands us to be careful in the selection of statistical material and in the application of statistics. If his warning were heeded, it would save us from much imported and domestic rubbish. Curiously enough, the fact is not mentioned that in the preparation of the mortality statistics of artificially fed infants it is of importance to know whether the purity of the cow's milk taken by the infants has been up to our modern requirements.

The chapter on "Milk" is written by Raudnitz, who treated the same subject in the "Ergebnisse der Physiologie" and whose collective abstracts on milk chemistry in the "Monatsschrift für Kinderheilkunde" are a welcome aid to anybody interested in this subject. We encounter the same positive assurance and sometimes the slight lack of clearness, with which we have become acquainted in other contributions of this author.

"The Female Breast" is from the pen of Engel, whose association with the "Säuglingsheim" in Dresden puts him in a good position to treat such subjects as "Ability to Nurse," "Technique of Nursing," "Hygiene of the Nursing Woman," "Choice of a Wetnurse," etc. Some of the single paragraphs are a little scanty, and a fuller discussion of the contraindications of nursing as well as of the drugs eliminated through the mammary glands should have found its place in these pages.

Camerer, the younger, contributes the chapter on "Metabolism and Nutrition During the First Year of Life." Following in the footsteps of the elder Camerer, a pioneer in this field of investigation, he lays the greatest stress on the energy metabolism of the infant. And while it is made very plausible that the food requirement of the infant is mainly determined by its energy metabolism, still the other aspects of metabolism like nitrogen and mineral metabolism, etc., are of much importance and should have received a little more attention.

In the paragraph on "Nutrition and Metabolism of the Breast-fed Infant During the First Week of Life," the author states that it is undesirable to give the breast more than eight times during 24 hours and recommends 6-7 feedings, while later on he recommends for the first three months the same number of feedings. This recommendation differs from that advocated on the basis of stupid tradition in most of our American text-books.

The paragraph on "Artificial Feeding of Infants" contains this passage: "A uniform method, which must be adopted in every case of artificial feeding, does not exist. This fact is frequently cited at present to prove our lack of sufficient knowledge, but it simply shows that an infant may be reared by different methods." In the presentation of the artificial feeding, the American reader will be surprised at the absence of the frightful array of formulas and charts, with which he is customarily confronted. And while a $\frac{dx}{dy}$ may well be adapted to create in the mind of the unsophisticated reader the impression of the author's profound learning, its intrinsic value for infant feeding is very problematical. The shortness, with which the technique of artificial feeding is described—it occupying not quite three pages—is enough to make one gasp with wonder mingled, we must confess, with some admiration. The two paragraphs contain much good sense and are singularly free from decorative embellishments.

In the chapter on "Children's Growth in Weight and Height," the same author points out that the weight of an infant during the first year and even later is dependent to a certain extent on its birth weight, a fact very frequently neglected, when some authors want to feed babies up to a certain average weight for a given period.

A chapter on "Nutrition After the First Year" by Sommerfeld is enriched with a chart giving in graphical form the average composition of the more important articles of food employed in childhood; and the chapter "Composition of Urine and Feces After the First Year of Life" concludes the first volume.

Volume II begins with "The Diseases of the Newborn," by Knöpfelmacher. Eighty pages are devoted to this subject. The three chapters following are written by Rommel: "Prematurity and Congenital Debility" with a discussion of the principles and construction of incubators and the technique of feeding their inmates; "Asphyxia and Atelectasis" and "Scleroderma and Sclerema."

The "Diseases of Puberty" are treated by Seitz in an attractive though cursory manner. The importance of the statement, "The physiological sexual development is a critical period, requiring much insight and consideration from parents and teachers" is well recognized by many physicians, but not sufficiently, we fear, by parents and by school authorities.

Under the heading "Constitutional Diseases," several chapters are included:

1. "Diseases of the Blood and of the Blood-preparing Organs," by Japha. A special paragraph of nearly five pages is devoted to the "Anæmia of School Children," and it contains much that is

of interest. Hodgkins' disease is included among the rather ill defined forms of pseudoleukemia, it is not regarded as a disease, *sui generis*. The newer American literature is not taken into consideration (Reed, Longcope, etc.). It is true that the presentation of diseases of the blood, etc., is difficult, and, therefore, it is not surprising that we find room for much improvement in this part. Perhaps it would not be amiss to include here a discussion of the bacteriology of the blood, if the editors do not decide to give it a separate chapter.

2. "Hæmorrhagic Affections," by Hecker. This chapter includes hæmophilia, the different forms of purpura, scorbutus, and paroxysmal hæmoglobinuria.

3. "Infantile Scurvy," by von Starck.

4. "Rachitis," by Stoltzner, a believer in phosphorus medication.

5. "Diabetes Mellitus," and 6. "Diabetes Insipidus," both by von Noorden, whose special fitness to treat these subjects need not be pointed out.

7. "Scrofula," by Galge. The author adheres to the old term "scrofula," because he is of the opinion that even the most ingenious attempt to separate the affections previously classified under that name have only contributed to increase the confusion. He accepts an hereditary predisposition, whatever that may be, and is inclined to regard the whole train of affections of tubercular nature.

The "Infectious Diseases" conclude the second volume. "Measles" are described by Moser and "Scarlet Fever" by von Pirquet and Schick. The illustration of the different symptoms by showing their occurrence in selected cases lends greatly to the value of this chapter. The differential diagnosis is given full consideration. The therapeutic measures recommended are based on personal observations, and this portion seems to be very satisfactory, although in one instance we cannot quite agree with the authors, and we should not like to dismiss offhand the administration of urotropin as a prophylactic measure against nephritis. The serum treatment is only used in severe cases, because large amounts are necessary, and these are likely to cause a more frequent occurrence of intense serum disease. "German Measles" and "Dukes' Fourth Disease" are described by Bókay, who is inclined to accept the latter as a special disease. Swoboda contributes the chapter on "Varicella," and Voigt that on "Vaccination." The chapter on "Diphtheria," by Trumpp is well written, and the translator's parenthesis calling attention to the desirability of large doses of anti-toxin is not out of place. After the description of "Mumps" by Moro, follows the chapter on "Typhoid Fever," by Fischl. The importance of the chronic carrier of bacilli in the spreading of the disease is not mentioned. The Brandt bath as a therapeutic measure is stigmatized as coarse and undesirable. The recommendation to strengthen the waning heart-power by bold alcoholic administrations lacks a sound physiological or pharmacological basis. The short chapter on "Dysentery," by Langer is followed by one on "Influenza," contributed by Spiegelberg, which gives, on the whole, equal satisfaction as the next chapter on "Whooping Cough," by Neurath. In the etiology of "Acute Articular Rheumatism," by Ibrahim, the newer work of the English investigators may have deserved notice. The more important forms of specific arthritides (*i. e.*, due to different microorganisms) are described briefly. The description of "Chronic Articular Rheumatism" seems to be rather unsatisfactory. The whole chapter on rheumatism needs revision. For instance, the tonsilar infections in their possible relation to rheumatic affections are dismissed in a rather arbitrary manner.

The next chapter is a treatise on "Syphilis." It is sufficient to mention the name of the author, Hochsinger, whose classical studies of this disease are well known.

The chapter on "Tuberculosis" is signed by Schlossmann. The author is of the opinion that a not insignificant number of cases are of hereditary origin, although this mode of infection is by no means the most important one. We find the following sentence printed in italics: "Finally we must repeat that a successful campaign against tuberculosis can be waged only if we prevent infection during childhood. For it is during this period of life in the great majority of cases that the infection takes place, although the disease may not become manifest until later in life."

We expected much valuable information with regard to the time of infection from vaccination according to the method of von Pirquet, and perhaps from the ocular reaction of Wolff-Eisner and Calmette, but both of these methods did not exist when this chapter was written. We must, therefore, confine ourselves to the mention of the further statement that milk does not play an important rôle in the genesis of the disease, as it is generally cooked previous to being used. The problem of the transmission of tuberculosis by means of milk deserves a little more attention in the next edition.

The third volume opens with the "Diseases of the Digestive System," and the first chapter is Moro's description of the "Diseases of the Mouth." There occurs a little lapse on the first page, where potassium sulphocya*nide* is given as a characteristic ingredient of the adult's saliva. It should read sulphocya*nate*. The syphilitic and tubercular lesions of the mouth should have received a passing attention, if no more. The so-called Riga's disease, it may be mentioned, has been observed in America, too. Difficult dentition as a morbid entity with all kinds of consequences is accepted with a readiness which should have been guarded in view of the violent storms of discussion through which this foster-child of the windy clinician has passed. "The Diseases of the Tonsils, Pharynx, and Œsophagus" are described by Finkelstein.

The "Diseases of Nutrition in Infancy," by Fischl, occupy about 89 pages. The title more commonly seen is gastro-intestinal disturbances, but, owing to the efforts of Czerny and Keller, the title given above gains ground steadily. In reviewing a work of the scope of the one before us, we must necessarily refrain from a detailed discussion of this chapter. The enormous variety of these ailments and their infinite transitions from one to the other render a satisfactory classification well-nigh impossible. A detailed classification will always arouse a great deal of dissension and a more general one, like that of the author, inasmuch as it does not share the same fate, will be of relatively little use to the practitioner. But we should expect in the presentation of this subject that the author would pass in review all the different factors, dietary, bacteriological, pathological, etc., which probably participate in the nosology. And of this task the author has acquitted himself on the whole very creditably. We must restrain from entering upon the discussion of the single groups and the therapeutic measures proposed, although we cannot subscribe to all the author gives us. But with regard to immunization therapy in cases where infants respond with severe signs of intoxication to even small amounts of cow's milk, we must recommend greater caution in the light of the researches of Anderson and Rosenau concerning anaphylaxis.

The "Local Diseases of the Stomach and Intestines in Earlier Childhood," as well as "The Gastro-intestinal Diseases of Older Children" come from the pen of the same author. The next edition will no doubt contain the new paraffin treatment of prolapsus recti.

The chapter on "Pyloric Stenosis in Infancy," by Pfaundler, gives a clear and concise picture of this disease.

Selter, writing on "The Diseases of the Appendix" does not advise immediate operation in every case of appendicitis or periappendicitis, but still concedes an important part to the expectant treatment. A consideration of Simon's septic factor would not have been amiss. The chapter on "Herniæ in Children," is by the same author, and Langer, considers the "Animal Parasites."

The "Diseases of the Peritoneum" and the "Diseases of the Liver" are described by Stoos.

In the next chapter Freund treats of the "Pathology of Metabolism." The main points known at the time of the writing are clearly stated by the author, who has contributed much original work to this subject. The reader must be impressed by the scarcity of valuable data at our disposal and also by the importance of this field of investigation. Anybody conversant with the American pediatric literature must feel keenly the utter neglect of this most important branch of pediatrics barring few exceptions, and that in spite of the fact that there exist in this country several well-appointed children's hospitals, where such researches could have been undertaken very readily. The advances in one of our most important branches—the establishment of infant feeding on a sound, scientific basis—*must* come through this channel. It is true, this work requires earnest and honest application, and it is a question of slow and cautious progress. The work of many hours will yield only relatively few printed pages and the results will not appeal to the masses. We hope sincerely that the next edition will be able to chronicle progress made on this side of the Atlantic.

In the next chapter, "Intestinal Bacteria," we encounter its author, Moro, in his own field.

The "Poisons" are considered by Schlossmann, while the "Diseases of the Nose, Trachea, Bronchi, Lungs, and Pleura" are described by Feer in an able manner. Gallatti contributes a brief chapter on the "Diseases of the Larynx." The "Diseases of the Thymus, Status Lymphaticus and Sudden Death in Infancy" we find over the signature of Friedjung. If the reader expects to find a detailed discussion of the status lymphaticus he will be disappointed, for it is only considered in connection with the explanation of sudden death associated with an enlargement of the thymus.

Hochsinger contributes the "Diseases of the Circulatory System." The author adheres to his assumption of the complete absence of accidental (so-called anæmic) heart murmurs during the first years of life, although he states that exceptions have been taken to this view.

The chapter on "Diseases of the Thyroid Gland" by Siegert, concludes the third volume. Here several affections are included which do not seem dependent on any demonstrable changes in the thyroid as infantile myxidiocy, mongolism, and micromelia. The parathyroids are not taken under consideration.

The fourth volume begins with the "Diseases of the Urogenital System," by Langstein. The statement that creatinin has not been demonstrated in the urine of healthy, milk-fed babies must be corrected. In the description of the different forms of nephritis more stress should be laid on the consecutive conditions of the heart. The subject is treated in 110 pages, and the omissions are few. It may be somewhat doubtful whether Addison's disease finds its proper place in this chapter.

The "Peculiarities of the Child's Nervous System" written by Pfister are followed by the "Organic Diseases of the Nervous System." Zappert has accorded to this subject in about 160 pages the careful treatment which might have been expected from this author. The name of Thiemich is so intimately connected with "Functional Diseases of the Nervous System" of childhood, that his signature under this title is not a matter of surprise. The usual forms of tetany, laryngospasm, and eclampsia infantum are considered as different manifestations of the spasmophile diathesis. Certain forms of laryngospasm and eclampsia infantum do not belong to this general group, and may be differentiated from the usual forms. In a separate paragraph the spasmophile diathesis is given as a clinical entity, and the influence of diet is pointed out. Its relation to the parathyroids is regarded as doubtful. Teething as a reflex cause of a convulsive attack on the basis of the spasmophile diathesis is absolutely denied. The efficiency of phosphorus and cod-liver oil in this condition, we may mention, may perhaps find its explanation in an influence on the calcium metabolism.

The whole presentation of the subject is eminently satisfactory. Epilepsy, neurasthenia, and hysteria pass before our view in a masterly manner. Some points may be open to discussion, but, as a whole, we doubt whether these subjects have ever been presented in such an attractive and interesting form, at least in the literature of pediatrics. The chapter on "Psychoses" suffers by comparison with the preceding chapters, and it deserves more space than four pages.

The same author contributes the "Diseases of the Meninges." We are rather surprised that the method of demonstrating the presence of tubercle bacilli in the sediment of centrifugalized cerebrospinal fluid is not mentioned. This method proved very successful in the hands of Bernstein in this country. The author concludes his description of meningococcus meningitis with these words: "Following the trend of the times a number of modern authors base their hopes upon a future serum therapy." We owe it to Flexner that this hope has, to a certain extent, found such a speedy fulfillment.

Galewsky devotes 89 pages to "The Most Important Diseases of the Skin." The American reader would have liked to find included the skin affections due to poisoning with Rhus toxicodendron.

With Leiner's description of "The Tuberculous Lesions of the Skin" the fourth and last volume finds its conclusion. Throughout the work numerous excellently executed illustrations and charts form a very attractive feature, and in many instances aid the understanding considerably.

We are aware that our review has been a rather cursory one. Here and there our criticism may have been too lenient and we may have failed to point out more or less important omissions, but, as a whole, we are afraid that we have sinned more in the opposite direction. We must express a pardonable envy, if we contemplate the many active workers who have left a distinct imprint on their fields of investigations, and who have lent their support to the editors. The list of authors does not include all the best names. The editors themselves, Pfaundler and Schlossmann, rank high in pediatrics.

We believe that the American editors, Shaw and La Fétra, had a twofold object in view when they undertook their labors; first, to introduce the American reader, who may have no access to the foreign literature, to the views governing pediatrics in the German-speaking countries; and, second, to stimulate investigation along the lines indicated in the work. The first object they have accomplished as much as lay in their power with the willing help of the many translators, who have acquitted themselves of their task with credit. Whether they will accomplish their second object, the future only can show. But if not all the signs are deceptive, it seems that the younger pediatricians of this country are ready to liberate themselves from the baneful influence of a few self-constituted authorities and to participate in the free development of pediatrics on the basis of sound scientific principles.

S. A.

Diseases of the Nervous System. By H. CAMPBELL THOMSON, M. D. (Lond.), F. R. C. P., etc. With 8 colored and 12 black-and-white plates, and 101 figures in the text. Price, $2.75. (*Chicago: W. T. Keener & Co., 1908.*)

Dr. Thomson states in his introduction that in this volume his constant aim has been "to bear in mind the principal difficulties which are apt to beset the student"; and he has succeeded in his object and has written a serviceable manual, which may be recommended without any restrictions to those beginning the study of nervous diseases. In a small octavo volume of less than 500 pages, the author has presented the subject in an attractive and

clear manner, so that his book makes an admirable introduction to the larger volumes on nervous diseases. The publishers are doing an excellent work in bringing out a number of first-class English works in agreeable form. R. N.

Meat and Food Inspection. By WM. ROBERTSON, M. D., D. P. H. F. P. S., etc. With Regulations Governing Meat Inspection in the United States. By MAXIMILIAN HERZOG, M. D. Price, $3.50. (*Chicago: W. T. Keener & Co., 1908.*)

In the first half of this book the author discusses certain questions concerning meat and food inspection which are of common interest to all inspectors, but the second half is devoted to the laws which govern this problem in Great Britain, and these are of interest only to a select body of students, who are anxious to know how this inspection is controlled abroad as well as at home. It is a pity that the author did not make two distinct volumes of his work, for thus it would have had a better sale, at least in this country. It is to be feared that but few will purchase the volume, because of its expense, and because so much of it is devoted to that side of the question which does not appeal to the general reader. It is none the less a book to be recommended to all inspectors, for Dr. Robertson has had a wide experience, and has presented his information on the subject in a clear and concise manner. Other larger and more comprehensive works on food inspection exist, but this is a good one, especially adapted to the inspector who has not had a thorough training, and to the general practitioner who is anxious to have some knowledge of this subject. It would be well if all our country practitioners had a volume as excellent as this one on their shelves, for they should be able to instruct the farmers on many points in regard to furnishing milk and food to the community. It is this large body of doctors who can do much to eliminate illness in the cities by teaching their patients in the country how to keep their stables and barns clean. To these doctors this volume will prove really serviceable. R. N.

Transactions of the National Association for the Study of Epilepsy and the Care and Treatment of Epileptics. Seventh Annual Meeting, Richmond, Va., October 24, 1907. Vol. V. Edited by WM. P. SPRATLING, M. D., Superintendent of the Craig Colony for Epileptics, etc.

The most notable paper in this volume is that by Dr. C. C. Southard, entitled "Observation of Histo-Pathology in Epilepsy, Submitted in Competition for the Prize of $500 Offered for the Best Essay Bearing upon the Etiology of Epilepsy, the Paper being Awarded Honorable Mention." The author's summary in brief is as follows: "The theory of epilepsy expounded in the present paper is founded mainly upon structural considerations. The histological data have been interpreted largely from a functional point of view. The theory lays claim to some originality in two directions, in setting forth, namely: the properties of a typical epileptogenic focus in the cerebral cortex and the nature of that change in cortical tissue which favors epileptic discharges." There are numerous briefer papers by other members of the association, dealing with certain distinctive features of epilepsy in a practical way. An important part of the volume is the "Report of the Public Care of Epileptics from States, Foreign Countries, and Municipalities." Like so many other associations this one suffers from lack of means to pursue certain courses which it is anxious to do, and yet there are few associations which deserve more general and generous support. Epilepsy, if not a national, is at least a State problem, and should be handled in a broad-minded way. Our legislators must be taught the present sufferings and needs of epileptics, and must learn that this unfortunate class cannot be left to take care of itself, and should not be roughly classed with the insane. It is a problem like that of tuberculosis, insanity, and venereal disease, which must be sooner or later solved, and the solution will only be reached by co-operation between the public and the medical profession. R. N.

Golden Rules of Dietetics. The General Principles and Empiric Knowledge of Human Nutrition; Analytic Tables of Foodstuffs; Diet Lists and Rules for Infant Feeding and for Feeding in Various Diseases. By A. L. BENEDICT, M. D. Price, $3.00. (*St. Louis: C. V. Mosby Medical Book and Publishing Company, 1908.*)

The author has failed to digest well all the information that he has collected, and the result is unsatisfactory. A more appropriate title for the book would have been "Dietetics"; it is impossible to discover what the "Golden Rules" are. It is unfortunate that so few of the medical profession can write lucid and grammatical English—there are many passages in this volume that suffer from the author's impossibility to express his meaning clearly, and according to the first principles of grammar. Had the author been willing to write a simple little hand-book on dietetics he might have produced a useful volume, but there are many books, no larger than this one, which deal far more satisfactorily with the subjects discussed. R. N.

STUDIES IN GENITO-URINARY SURGERY

THE JOHNS HOPKINS HOSPITAL REPORTS, Volumes XIII and XIV, now ready. Volume XIII consists of about 605 pages with 201 figures in the text, six plates and one colored chart.

Volume XIV consists of about 632 pages, and has 97 figures in the text.

STUDIES IN PNEUMONIA

In press. Volume XV.

The price of each volume is five dollars ($5.00), bound in paper, and five dollars and fifty cents ($5.50), bound in cloth.

Remittances and orders will now be received.

THE JOHNS HOPKINS PRESS, BALTIMORE, MD.

The Johns Hopkins Hospital Bulletins are issued monthly. They are printed by the LORD BALTIMORE PRESS, Baltimore. Subscriptions, $2.00 a year (foreign postage, 50 cents), may be addressed to the publishers, THE JOHNS HOPKINS PRESS, BALTIMORE; single copies will be sent by mail for twenty-five cents each. Single copies may also be procured from the BALTIMORE NEWS CO., Baltimore.